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A BACTERIOLOGIC STUDY OF PENETRATING WOUNDS OF THE BRAIN, FROM THE SURGICAL POINT OF VIEW

MAJOR ARTHUR D. ECKER, M.C., A.U.S.*

(Received for publication June 20, 1945)

IN THE SUMMER of 1944 there became available for the first time a series of penetrating wounds of the brain due to shell fragments in which the patients had been treated with both sulfadiazine and systemically administered penicillin. This study (Table 1) is based on 50 such wounds, some of which had not been touched whereas others had received preliminary operations at forward hospitals.

There were 42 cases in which bacteriologic studies of the *brain* were made, either at operation or on admission to the hospital. The wounds were incurred from 3 to 86 days before they were studied, namely, between 11 June 1944 and 26 October 1944. Almost all the wounds were caused by shell fragments. There were retained bone fragments within the brain in almost every case. Smears and cultures were made by Lt. Harry Blechman and doubtful organisms were checked by the 1st General Medical Laboratory. The swabs were bacteriologically sterile in 10 cases, in all of which the scalp had been previously sutured. In the other 32 cases the organisms cultured from the brain were as follows: Staphylococcus aureus, 7 cases; Staphylococcus albus, 17; Streptococcus viridans, 9; non-hemolytic streptococcus, 9; gram-negative bacilli, 6; Micrococcus tetragenus, 4; and Clostridium, 2 cases. Surprisingly, there were no hemolytic streptococci in this series. The gram-negative bacilli were always found in association with coccus forms.

From the study of the x-ray films and findings at operation an attempt was made to estimate the number of cubic centimeters of brain destruction in each case. The figures range from 5 to 250 cc. However, this estimated volume of brain tissue destroyed had no apparent bearing on whether or not there were viable bacteria present in the wound.

There were 32 cases in which separate cultures were made of the superficial and of the deeper layers of the brain wound. In most of these cases the same organisms were cultivated from each layer of the wound. However, there were 3 where the superficial tissues of the brain were sterile but the deep tissues contaminated. Conversely, in 5 cases the superficial brain tissues were contaminated but the deep tissues of the wound were sterile.

Finally, there were 11 cases of wounds previously unoperated on, in which cultures were made at all three levels, scalp or skull, superficial brain, and deep brain. In 9 of these the organisms at all three levels were identical. The wounds underwent thorough debridement of the brain and watertight closure of the dura with a graft of pericranium or of temporal fascia and were

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closed without drainage. Adequate x-ray and surgical facilities, blood transfusions and drugs were available. Definitive operation in all of these cases, except 2, was carried out.

GRAM-NEGATIVE ORGANISMS

In the complete series of 50 brain wounds with dural penetration there were 3 deaths from infection, and in all 3 cases gram-negative bacilli were cultured from the brain prior to the patient's death. Cairns[1] had previously directed attention to the growing significance of gram-negative organisms in the brain since the advent of penicillin. The present author had found urea useful as an adjunct to the sulfonamide treatment in a case of B. coli meningitis.[2] Urea was used in the 7 subsequent cases of brain wounds contaminated by both cocci and gram-negative bacilli. The latter were variously described by different laboratories and the terms used included B. coli, coliform group, paracolon, Klebsiella pneumoniae and Aerobacter aerogenes. In addition to the routine surgical treatment and administration of penicillin and sulfadiazine, urea was added in dosage of 30 grams every four hours by mouth. None of these patients died.

CLOSTRIDIAL CONTAMINATION

There were 4 cases of clostridial contamination of the brain, 2 of which are included in Table 1 and in all of which the patient recovered.

Example. This soldier was wounded in action in France on 25 July 1944, by shell fragments which penetrated the left temporal lobe of the brain. At an evacuation hospital a portion of the brain tissue, 3 cm. in diameter, was debrided and a drain was left in place. The patient was in critical condition when placed under the author's care on 31 July 1944. There was a brain fungus, about 4 cm. in diameter, which was necrotic and was surrounded by profuse pink discharge containing bubbles, and which yielded a foul odor. Clostridium Welchii and Clostridium putrificum as well as Aerobacter aerogenes, hemolytic Staphylococcus aureus and Streptococcus faecalis were cultured from the wound. The patient was given supportive therapy including transfusions of whole blood, penicillin, sulfadiazine and a small amount of gas gangrene antitoxin. On 7 August 1944 another herniated portion of the brain was removed digitally without anesthesia. On 23 August 1944 cerebrospinal fluid suddenly began to gush from one of the two wounds; the brain was obviously under increased tension. Accordingly, the following emergency surgical procedure was undertaken.

The original scalp flap was turned back and revealed a large cerebral fungus from which cerebrospinal fluid issued. There were multiple brain abscesses in the temporal lobe extending in all directions from the wound. There were six discrete pockets of thin, yellow pus. The brain was a veritable honeycomb of fibrous tissue and purulent material. The fibrous tissue was 1 cm. thick in places. The largest individual abscess contained about 10 cc. of pus. Obviously necrotic brain, which was removed, amounted to approximately 150 gm. or a spherical mass 7 cm. in diameter. The left lateral ventricle was opened widely and some of the choroid plexus was removed. The temporal fascia from the anterior portion of the temporal fossa was rotated so that a careful watertight closure of the dural defect was effected. The scalp flap had a very poor blood supply because of its shape, and the incision for drainage at its base. Penicillin, sulfadiazine and urea were administered.

Cultures were made from 4 of the abscess cavities, each of which yielded E. coli and a non-hemolytic anaerobic streptococcus. In addition, the swab from one abscess cavity yielded Clostridium sporogenes. The patient gradually improved and began to say a few words by 10 September 1944. At the time of discharge from the hospital on 30 September 1944 the wound was practically healed.

DISCUSSION

It is clear that systemically administered penicillin and sulfonamide act as bacteriostatic agents on penetrating wounds of the brain just as they do in wounds of other parts of the body. That is, they keep the organisms that are driven into the depths of the brain from infecting the brain tissues. It is impossible to judge on bacteriologic and histologic grounds alone when such bacterial contamination becomes an infection. In non-traumatic cases necrosis indicates that the presence of the organisms in the tissue is destructive, and that, therefore, an infection is present. Such a criterion is, of course, of no value in surgical cases because traumatic necrosis is always present. Perhaps the change from contamination to infection is best expressed in biological terms. For example, if the contaminating organisms thrive and multiply to the detriment of the host, an infection may be considered to be present.

In one case, where there had been some drainage from the wound following an earlier partial debridement, an ordinary swab of the tract in the brain was sterile but the biopsy specimen of the wall of the tract yielded organisms.

Furthermore, there were 2 cases of retained bone fragments where there was no clinical evidence of infection and yet viable organisms were present in the brain. A good scalp closure had been performed in a forward hospital, both patients seemed quite well and there was neither stiff neck nor any other clinical evidence of infection, although there were several retained bone fragments in the brain. The spinal fluid was normal in every respect and even air encephalograms revealed no evidence of inflammation. Such latent infections may cause brain abscess in the future. It seems advisable to recommend a total excision not only of the retained bone fragments, but also of all surrounding gliotic tissue.

CONCLUSIONS

Bacteriologic studies of brain wounds due to shell fragments in the Normandy campaign revealed the organisms usually found in soil and on the human skin. The routine use of penicillin and sulfadiazine systemically maintained these bacteria in a state of contamination, rather than that of infection, for at least several days. Thorough debridement of the brain and watertight closure of the dura[3] were carried out in almost all cases with satisfactory results. Under such management the most dangerous organisms proved to be the gram-negative bacilli, which, however, were controlled when large doses of urea were administered as an adjunct to sulfonamide therapy.

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TABLE 1

Number	Date of Injury	Location	Estimated ec. Brain Destruction	Operation Here	Days from Injury to Def. Surgery	State of Scalp at Def. Operation	Bone Fragments in Brain		Bacterial Cultures			Urea Used	Infections	Days after Operation Patient Left	Condition at Time of Leaving
							Before Def. Operation	After Def. Operation	Scalp and Skull	Superficial Brain	Deep Brain				
1	26 May 44	Ant. Fossa	16	Prim.	2	Open	Yes	No	IINH			No	None	50	Well
2	28 May 44	Ant. Fossa	2	Prim.	6	Healing	Yes	No				No	None	13	Well
3	25 May 44	Ant. Fossa	8	Prim.	12	Healing	Yes	No	IINH IIIH			No	None	39	Well
4	8 June 44	Occipital	14	Prim.	3	Open	Yes	No	IINH IIINH		IIH IIINH	No	None	23	Well
5	10 June 44	Frontal	28	Sec.	3	Healing	Yes	No	IH IINH			No	Fungus	29	Dead
6	8 June 44	Frontal	4	Prim.	13	Open	Yes	No	IINH			No	None	58	Well
7	6 June 44	Ant. Fossa	18	Prim.	9	Healing	Yes	No		IINH Mt	IINH Mt	No	None	32	Well
8	10 June 44	Frontal	5	Prim.	5	Open	Yes	No	IINH	Sterile	IINH	No	None	30	Well
9	11 June 44	Parietal	10	Prim.	5	Healing	Yes	No	IINH			No	None	49	Well
10	6 June 44	Temporal	120	Sec.	11	Healing	Yes	No	IINH IIINH	Sterile	Sterile	No	None	21	Recovering
11	9 June 44	Temporal	19	Sec.	14	Healing	No	No	Sterile		Sterile	No	Superf. Scalp	30	Recovering
12	10 June 44	Ant. Fossa	15	Sec.	15	Open	Yes	No	Sterile		Sterile	No	None	21	Well
13	11 June 44	Post. Fossa	6	Prim.	16	Healing	Yes	No	IH	IH IINH	IH INH	No	Superf. Scalp	29	Superf. scalp Infection
14	6 June 44	Ant. Fossa	2	Prim.	18	Healing	No	No			Sterile	No	None	32	Well
15	14 June 44	Ant. Fossa	16	Sec.	23	Healing	Yes	No	IIIV E. coli	IIIV E. coli	IIIV E. coli	Yes	Meningitis	78	Post-meningitic hydrocephalus
16	16 July 44	Parietal	25	Prim.	5	Open	Yes	No	IINH IIIV Aa ClW	IINH IIIV Aa ClW	IINH IIIV Aa ClW	Yes	Superf. Scalp	28	Recovering
17	10 July 44	Temporal	35	Sec.	12	Healing	Yes	No	IIIV IIINH Mt	IIIV	IIIV	No	Superf. Scalp	56	Well

Number	Date of Injury	Location	Estimated ec. Brain Destruction	Operation Here	Days from Injury to Def. Surgery	State of Scalp at Def. Operation	Bone Fragments in Brain		Bacterial Cultures			Urea Used	Infections	Days after Operation Patient Left	Condition at Time of Leaving
							Before Def. Operation	After Def. Operation	Scalp and Skull	Superficial Brain	Deep Brain				
18	11 July 44	Temporal	130	Sec.	12	Open	No	No	IH IIINH Aa	IIIV	IIIV	Yes	Encephalo-meningitis	16	Dead
19	23 June 44	Ant. Fossa	56	Sec.	32	Healed	Yes	No		IIINH Mt	II IIINH	No	None	53	Well
20	13 July 44	Temporal	160	Sec.	13	Open	Yes	No	IH IIINH Aa Mt	IIINH	IIINH Mt	No	Superf. Scalp	57	Well
21	13 July 44	Occipital	60	Sec.	15	Draining	Yes	No	IH IIINH	IIIV	IIIV	Yes	Superf. Scalp	49	Well
22	13 July 44	Ant. Fossa	50	Sec.	21	Healed	Yes	No		Sterile	Sterile	No	Superf. Scalp	25	Well
23	24 July 44	Parietal	60	Sec.	17	Healing	Yes	No	IINH	Sterile	Sterile	No	Superf. Scalp	41	Well
24	1 Aug. 44	Post. Fossa	27	Prim.	7	Healing	Yes	No	INH IIINH Mt		IH IIINH Mt	No	None	38	Well
25	25 July 44	Temporal	21	Sec.	13	Healing	Yes	No	IH IIINH	IH IIIV	Sterile	No	None	37	Well
26	31 July 44	Ant. Fossa	40	Prim.	10	Healing	Yes	No	Sterile		Sterile	No	None	37	Sl. mental dullness
27	9 Aug. 44	Parietal	45	Prim.	5	Open	Yes	No	IIINH Mt	IIINH Mt	IIINH Mt	No	Superf. Scalp	14	Wound granulating
28	9 July 44	Parietal	48	Sec.	19	Draining	Yes	No	IH IIINH Kp	IIIV	IIINH Kp	Yes	Encephalo-meningitis	26	Draining wound
29	11 Aug. 44	Frontal	100	Sec.	6	Open	Yes	No	Kp	Kp	IINH	Yes	Superf. Scalp	37	Granulating
30	26 July 44	Occipital	120	Prim.	30	Healed	Yes	No	Sterile		IINH	No	None	31	Well
31	25 July 44	Temporal	160	Sec.	29	Open	No	No	IH Aa ClW III	IH E. coli	IH E. coli ClW	Yes	Brain abscess	37	Granulating
32	26 July 44	Frontal	8	Sec.	35	Open	No	No		IH IIINH	Sterile	Yes	None	31	Well
33	27 Aug. 44	Parietal	200	Sec.	15	Draining	Yes	No		IH IIIV Kp	IH IIIV Kp	Yes	Superf. Scalp	40	Small defect
34	28 Aug. 44	Ant. Fossa	15	Prim.	9	Healed	Yes	No		IINH	IINH	No	None	23	Well

TABLE 1—*Continued*

Number	Date of Injury	Location	Estimated cc. Brain Destruction	Operation Here	Days from Injury to Def. Surgery	State of Scalp at Def. Operation	Bone Fragments in Brain		Bacterial Cultures			Urea Used	Infections	Days after Operation Patient Left	Condition at Time of Leaving
							Before Def. Operation	After Def. Operation	Scalp and Skull	Superficial Brain	Deep Brain				
35	9 Aug. 44	Parietal	80	Sec.	29	Healed	Yes	No		IIH IIINH	IIH IIINH	No	None	30	Well
36	13 Aug. 44	Occipital	120	Sec.	15	Draining	Yes	No	Mt	Sterile	IINH IIIV	No	None	35	Well
37	14 July 44	Temporal	25	Sec.	57	Healing	Yes	No	IIH IINH	IIH	Sterile	No	None	32	Well
38	8 Sept. 44	Occipital	30	Prim.	3	Open	Yes	No	IIH	IIH	IIH	No	None	22	Well
39	31 Aug. 44	Parietal	200	Sec.	10	Healing	Yes	No	IIH IIINH	IINH		No	None	1	Dead
40	5 Sept. 44	Parietal	240	Sec.	10	Open	No	No		IINH Bs	Sterile	No	None	20	Wound granulating
41	2 Sept. 44	Occipital	110	Sec.	11	Healed	Yes	No		Sterile	Sterile	No	None	28	Well
42	3 Sept. 44	Interventricular.	75	Sec.	11	Healing	Yes	No	IH IINH	Sterile	IIH	No	None	30	Well
43	7 Sept. 44	Bi-occipital.	75	Sec.	9	Draining	Yes	No		Sterile		No	None	33	Well
44	14 Sept. 44	Temporal	10	Sec.	5	Healing	Yes	No	IH IINH	Sterile	Sterile	No	None	2	Dead
45	12 Sept. 44	Ant. Fossa	20	Prim.	7	Open	Yes	No	Sterile			No	None	16	Well
46	9 Sept. 44	Frontal	12	Sec.	6	Healing	Yes	No	IINH IIINH	IIH	IIH	No	None	32	Well
47	6 June 44	Frontal	120	Sec.	20	Draining	Yes	No	IINH IIINH	IINH	Sterile	No	Encephalo-meningitis	27	Wound granulating
48	23 June 44	Frontal	250	None		Draining	No			IH IIINH	IH IIINH	No	Encephalo-meningitis		Dead
49	21 June 44	Frontal	150	None		Draining	Yes					No	Encephalo-meningitis		Dead
50	30 July 44	Frontal	60	Sec.	86	Healed	Yes	No		IH	IH	No	None	22	Well

CODE

I Staphylococcus aureus
II Staphylococcus albus
III Streptococcus
H Hemolytic
NH Non-hemolytic
Aa Aerobacter aerogenes
Mt Micrococcus tetragenus
ClW Clostridium Welchii
Kp Klebsiella pneumoniae
V Viridans
Bs Bacillus subtilis

OBSERVATIONS ON EARLY TYPE OF BRAIN ABSCESS FOLLOWING PENETRATING WOUNDS OF THE BRAIN

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IN THE COURSE of the postoperative care, at the 36th General Hospital, of 206 penetrating wounds of the brain, due to shell fragment or bullet injuries, brain abscess occurred in 33 cases (16 per cent). Brief observations concerning the diagnosis, surgical treatment and chemotherapy are made in view of the commonness of this complication and, in many instances, the difficult problem of management.

MATERIAL

In this report the term "brain abscess" is used to signify a purulent collection or collections within the brain substance, characteristic of all the lesions. An expression such as "purulent encephalitis" might be used. In all cases, the cerebral substance had undergone a sufficient degree of necrosis to form frank pus in a cavity or in cavities. Since the collections were deep seated, Steinthal's term, "cortical ulcer," does not apply. The abscesses occurred early; they were associated with wound infection and frequently with a hernia or fungus. With but few exceptions they were not grossly encapsulated but were localized. Denser inflammatory tissue frequently bordered the collections. There were multiple loculated areas of pus in some instances in contrast to a single collection in others. Meningitis was also associated with the localized infection in some of the patients. The abscesses were located at the sites of the original debridements and usually also in the tracts produced by the metallic or bone fragments.

Primary debridements had been performed at American evacuation hospitals upon all patients except two. Of the group of 33 cases presented, 10 abscesses occurred among German patients and eight of these were treated at a Prisoner of War Hospital under difficult conditions. Three of the latter group were operated upon by Captain W. Klemperer and one by Captain T. Weaver. Two of the American patients had drainages of the abscesses performed while in the evacuation hospitals by Major W. Pitts and Captain E. Shearburn.

DIAGNOSIS

Two considerations appear to be important in the diagnosis of a complicating brain abscess. First, the presence of an infected wound; second, the presence of retained bone fragments in the brain following primary debride-

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Kp Klebsiella pneumoniae
V Viridans
Bs Bacillus subtilis
II Staphylococcus albus
III Streptococcus
H Hemolytic
NH Non-hemolytic
Aa Aerobacter aerogenes

ment. Only three patients presented healed wounds with an underlying purulent abscess. All others had infected wounds, which failed to heal with or without herniae or fungi. Four cases of superficial cortical necrosis ("cortical ulcer") associated with purulent wound infections have not been included in this series.

The presence of bone fragments in a subcortical tract after primary debridement of a penetrating wound is presumptive evidence for a brain

TABLE 1

Time from wounding to primary operation of of the group

Under 10 hours	5
10–15 hours	4
16–24 hours	6
24–48 hours	11
Over 48 hours	4
Unknown (over 24 hours)	3

TABLE 2

Time of abscess drainage from time of injury

6–10 days	5
11–15 days	6
16–20 days	10
20–25 days	12

TABLE 3

Locations of the abscesses (right 12—left 21)

Right frontal (1 death)	4	Left frontal	3
Right parietal	5	Left temporal	1
Right parieto-occipital	1	Left parietal (1 death)	7
Right frontoparietal—massive	1	Left parieto-occipital	2
Right fronto-temporo-occipital—hemispheric (death)	1	Left temporoparietal	1
		Left frontoparietal (1 death)	1
		Left occipital	1
		Left parietal and right occipital—bilateral (death)	1
Left hemispheric—gutter (death)	1	Left parietal and left temporal—two abscesses (death)	1
Left frontal and right temporal—bilateral (death)	1	Left parietal and occipital (death)	1

abscess. In only one instance following secondary debridement for the removal of bone fragments were these found to be sterile. Bone, hair, helmet-liner material or other debris provided the locus for an inflammatory collection. In our experience, ragged metal fragments were usually laden with organic material. The size of a metallic fragment was of significance in terms of the tract of damage produced; its irregularity was also of importance because of its ability to carry contaminating material.

Retained bone fragments were present in 19 of the patients with abscess; metallic foreign bodies were present in 12. Delineation of bone fragments was dependent upon satisfactory roentgenograms. In several cases, in which the diagnosis was in doubt, encephalograms were performed.

SURGICAL MANAGEMENT

Several methods were used in the surgical treatment of brain abscess following complete debridement of the wound and the underlying abscess.

These were: (1) Closure of the scalp. (2) Closure of the scalp about a tube for the instillation of penicillin. (3) Exteriorization of the cavity.[1,3] (4) Exteriorization with a temporary dural graft (Figs. 1, 2, 3, 4, 5, 6, and 7).

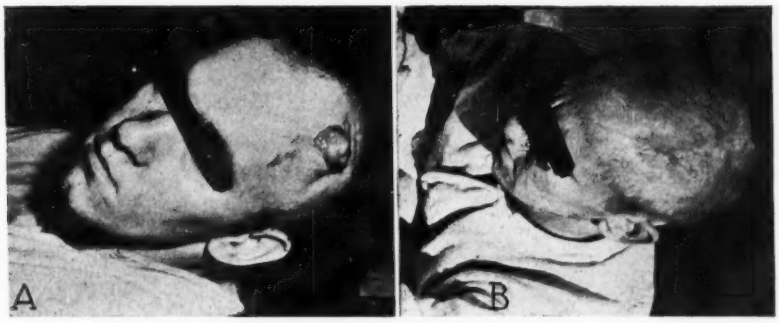

FIG. 1. Case 12. (A) Abscess treated by exteriorizing, with temporary herniation resulting. (B) Patient at time of evacuation with healed wound not requiring a skin graft.

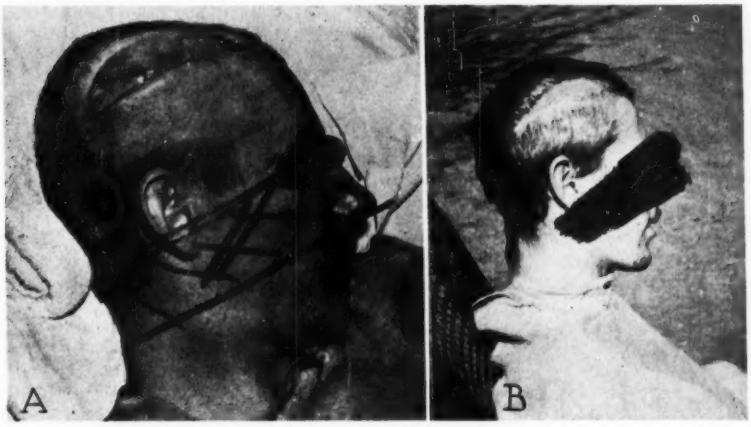

FIG. 2. Case 5. (A) Abscess treated by exteriorizing, with mild temporary herniation resulting. (B) Patient at time of evacuation with wound healing following application of a split thickness graft to conserve scalp for later cranioplasty.

From observations made in the various methods of treatment certain conclusions seem possible. One is that an early abscess following a penetrating wound of the brain must be treated by complete removal or debridement and not by "drainage." A second is that following debridement, no single technic for the wound management can be laid down as a uniform, ideal method. It would be desirable to close the wound tightly after wound excision and complete debridement of the abscess by suture of the scalp as has been proposed by Schwartz.[4] Figs. 4 and 5 are examples of this type of

treatment. There are situations where this is possible. In late purulent and non-purulent undebrided wounds, primary closure after thorough debridement has been successfully practised with and without the local use of penicillin and with or without a tube type of drain for the instillation of

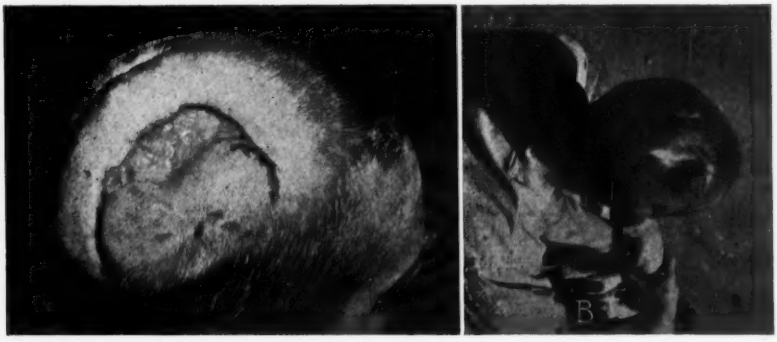

FIG. 3. Case 7. (A) Abscess treated by exteriorizing. Infection well controlled; mild herniation present. (B) Patient at time of evacuation with healed wound following application of a split thickness graft to conserve scalp for a later cranioplasty.

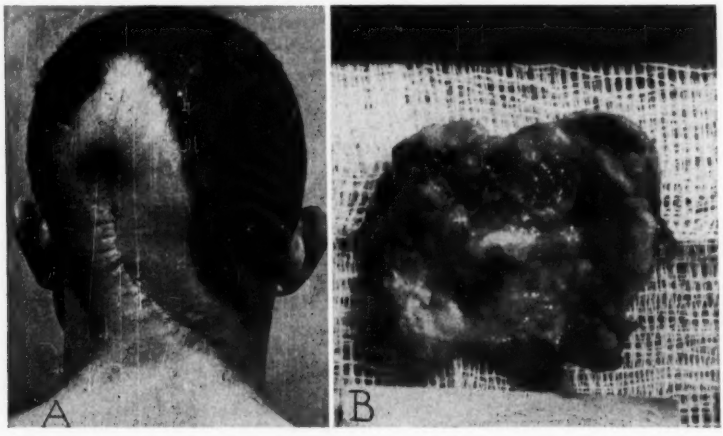

FIG. 4. Case 30. (A) Left occipital lobe abscess associated with an infected cervical wound. Excision of wound and complete enucleation of an encapsulated abscess. Primary wound healing followed. (B) Portion of the wall of an encapsulated abscess removed completely at secondary debridement.

penicillin.[2] This has proved to be a safe method. In the treatment of brain abscess, the technic of wound closure with the local instillation of penicillin via a small rubber tube has been successfully applied by English surgeons.[2] In our experience it has been difficult to either aspirate through the rubber tube or inject the penicillin. Seven patients were treated in this manner. The

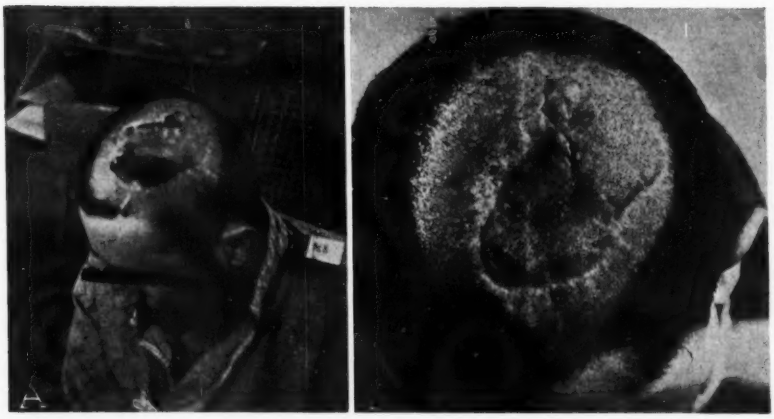

FIG. 5. (A) Patient at time of admission presenting an infected wound partially covered by a full thickness graft. An underlying brain abscess was present. (B) Appearance after debridement of scalp and abscess and closure of the scalp by mobilization. A dural graft was not used. Local instillation of penicillin through scalp.

FIG. 6. Case 24. Left parietal lobe abscess with meningitis treated by re-debridement and application of a temporary fascia lata graft to dural defect and subsequent split thickness graft.

rubber tubes, however, were removed in 48 hours and the penicillin solution injected through the scalp into the cavity of the debridement.

There are occasions, however, when these technics cannot be applied. In the presence of an infected wound with edematous and necrotic scalp edges and a large area of scalp loss; in situations where the debridement of

the abscess may not be as complete as desired; in abscesses involving the fronto-orbital-sinus area, with the threat of rhinorrhea, and the mastoid region, with otorrhea, primary closure of the scalp after debridement of the abscess would seem to be unwise and may be impossible.

In order to meet these conditions a modified "open" or "exteriorizing" method of treatment was used with success. Following scalp, pericranial, bone and dural revision, the abscess was debrided thoroughly and the cavity was then covered, temporarily, by means of a graft of fascia lata sutured to the debrided dural edge. Penicillin solution may be injected through the graft into the cavity. The temporary graft and bone were covered with vaseline gauze continuously. The graft was then removed at the end of a 15–20 day period and the underlying brain was prepared for a split thickness graft by means of moist boric compresses, since the cavity was not clean. The graft was applied without disturbing the scalp in order to allow a maximum of tissue for later cranioplasty.

FIG. 7. Occipito-mastoid wound complicated by otorrhea, meningitis and cortical ulceration. Wound was healed. Treatment by re-debridement and mastoidectomy with application of a fascia lata graft to dural opening; healing by granulation.

The temporary dural graft may serve several purposes. First, it prevents cerebral herniation if cerebral and meningeal infection is not under chemotherapeutic control. We have noted, following a penetrating wound with brain tissue loss, that even in a short period of time, within 20 days, the ipsilateral ventricle dilates and may be entered and soiled in the process of debridement of the abscess. This may result in meningitis with cerebral swelling, herniation and even fungus formation if the cavity has been "exteriorized" without a covering. In the technic of abscess debridement with scalp closure, extrusion of brain beneath the scalp may occur to a disadvantage. Second, in wounds that should not or cannot be closed, a temporary dural covering prevents cerebrospinal fluid leaking. A small communication with the ventricle closes readily by the early cerebral swelling in the area of the debridement. A large communication, however, closes after a period of delay as the brain fills the area of tissue loss. In this interval a dural covering controls the leaking. Frontal lobe abscesses communicating with the frontal or ethmoidal sinuses present the problem, particularly, of preventing rhinorrhea, accomplished by means of a graft which seals off the sinus orifices and the ventricular communication.

CHEMOTHERAPEUTIC MANAGEMENT

Two policies of drug therapy were used in the treatment of this group of patients. The first 15 were treated by sulfadiazine, penicillin being used only when sulfadiazine failed to control the infection. The following 18 patients were, in general, treated with penicillin. Sulfadiazine was used when the brain or spinal fluid cultures showed infection by gram negative organisms. There were several cases in which both drugs were administered. On the basis of this experience, the following routine was selected in the winter of 1944 for the chemotherapeutic management of cases of penetrating wounds of the brain complicated by infection. The drug was first administered that was easiest to give and that would exert the promptest effect. This was sulfadiazine. The patient's blood status was determined by the copper sulfate method and normal values were attained as rapidly as possible. Hydration was also speedily accomplished. Sulfadiazine (2 gm. every 4 hours, making a total of 12 gm. in 24 hours) was administered regularly for a period of five days with an equal dose of sodium bicarbonate. A daily blood count and urinalysis were recorded and an accurate record was made of the intake and output of fluids. If, at the end of that period, there was no clinical improvement; if the temperature was not decreased; or if the spinal fluid cell counts and protein were not lowered, penicillin therapy was begun: 50,000 units were administered intravenously immediately and then 25,000 units intramuscularly every three hours thereafter for two weeks; 10,000 units were given intraventricularly or intracisternally twice daily for four days in an attempt to sterilize the cerebrospinal fluid. In this interval of chemotherapy, if a cerebral abscess were diagnosed, it was evacuated as soon as possible and the drug of choice continued, based upon cultures made at operation and the type of surgical technic used in management. During the postoperative period, the blood chemistry was kept within normal limits; the caloric and vitamin intake was made adequate and the best possible nursing care was sought. That chemotherapy was merely an adjunct to thorough surgery was, of course, held as fundamental in the plan of management. It was recognized also that methods of employing chemotherapeutic agents were constantly subject to modification as knowledge and experience broadened.

RESULTS

The essential data for evaluating the results of treatment are lacking in this group of patients. The late complications that may occur and the final neurologic disability are unknown. A brief summary in terms of the mortality, which was 27 per cent, is possible, as is shown in Table 4.

An appraisal of results based upon the type of chemotherapy employed cannot be fairly made, since the number of cases is small. Among 15 patients treated with sulfadiazine, 4 deaths occurred. Penicillin also failed to control these fatal infections when used after the inefficacy of sulfadiazine was apparent.

Among 18 patients who were treated mainly by penicillin, 5 deaths resulted. Three of the 18 cases were transferred to sulfadiazine therapy when penicillin failed, all with favorable outcome.

A gram negative rod was the bacterium that caused the most difficult infection to combat with either sulfadiazine or penicillin, 3 deaths being caused by uncontrollable infection with this organism.

TABLE 4

Mortality in the surgical methods applied

Method	Cases	Mortality
I. Scalp closure	5	0
(Three cases were failures. Two were later exteriorized; a permanent dural graft had been used in one of these. The third was finally closed after failure of a first closure and failure of a second closure with a permanent dural graft.)		
II. Scalp closure about tube for instillation of penicillin	7	2
(One case was unsatisfactory, requiring reoperation with treatment by a temporary dural graft.)		
III. Simple exteriorization	10	5
(One case which was exteriorized was reoperated upon and a temporary dural graft applied.)		
IV. Exteriorization with use of a temporary dural graft	11	2

SUMMARY

1. Thirty-three cases are presented of early cerebral abscess complicating penetrating wounds of the brain.

2. The characteristics and diagnosis of these lesions are briefly considered and certain surgical and chemotherapeutic considerations discussed.

3. The abscesses were managed by several methods. Following complete debridement, which was held to be essential, a number were treated by a modified method of exteriorizing with a temporary dural graft covering of the cavity. After removal of the graft the wounds were then allowed to heal by granulation followed by split thickness grafting, to preserve the scalp for future cranioplasty.

4. Sulfadiazine was observed to be a highly effective drug adjunct to thorough surgical debridement of the abscessed area. Penicillin was used when sulfadiazine failed to control the infection.

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EYE SIGNS IN PINEAL TUMORS

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TUMORS of the pineal body are rare. According to Cushing's[1] figures, 0.7 per cent of his 2,000 intracranial tumors were pineal in origin. In spite of their rarity, however, certain clinical manifestations of growths in this region are of more than usual interest. Among these features are the disturbances concerned with pupillary activity, movements of the eyeballs and other ocular disorders. The paper here presented is devoted chiefly to a clinical study of these oculomotor and other related disturbances.

The material from which this information has been gathered consisted of 16 cases in which a diagnosis of pineal tumor was made at the Lahey Clinic during the years 1933 to 1944 inclusive. In 11 of the 16 patients the lesion was verified histologically either from tissue removed at operation or at autopsy. In the other 5 patients a presumed and almost certain diagnosis was made on the basis of the clinical evidence together with the finding of the characteristic filling defect in the posterior portion of the third ventricle as seen in the ventriculogram. These diagnostic features were supported by the demonstration of internal hydrocephalus and the complete relief of symptoms and signs following a decompression and roentgen therapy. In spite of the fact that it was not possible to verify the tumor histologically in these 5 patients, we feel that the clinical picture, the ventriculogram and the course following decompression and radiation therapy all support the diagnosis of pineal tumor with sufficient certainty to include them in this survey.

The surgical removal of a pineal tumor is an extremely difficult task and entails considerable risk. On the other hand, as Horrax and Daniels[5] have pointed out, subtemporal decompression followed by radiation therapy offers a comparatively safe method of treatment which has proved highly satisfactory in several cases. For this reason it is highly important to obtain all available evidence bearing upon the diagnosis of a growth in the pineal region. The oculomotor and pupillary disturbances furnish considerable information along these lines.

Before taking up in detail the various ocular disorders that may occur with pineal tumors, it may be well to note briefly certain general features shown by patients harboring these lesions. By recalling the strategic position of the epiphysis just above the corpora quadrigemina (Fig. 1), one can predict with a good deal of accuracy the most frequent signs and symptoms resulting from a tumor of this structure:

1. Increased intracranial pressure, often acute, producing headache,

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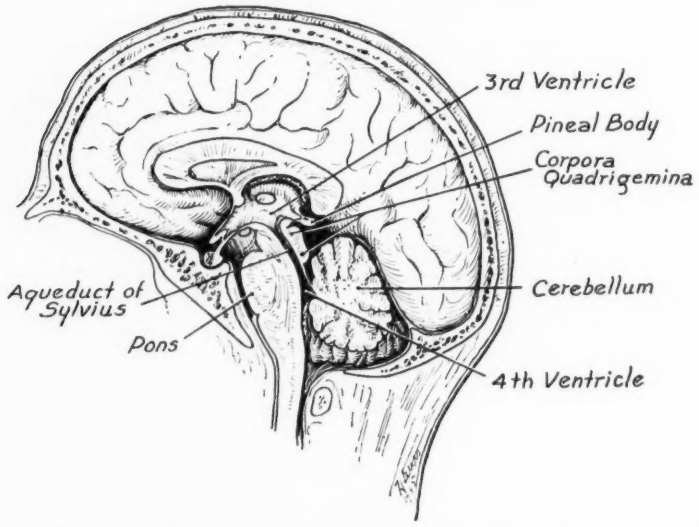

FIG. 1. Sagittal section of the brain showing the position of the pineal body, the corpora quadrigemina, aqueduct of Sylvius and third ventricle.

nausea, vomiting and papilledema. Mechanism: compression and obstruction of the aqueduct of Sylvius with resulting internal hydrocephalus.

2. Pressure on the corpora quadrigemina, giving rise to:
   (a) Eye signs.
      (1) Impaired pupillary reactions.
      (2) Limitations of extra-ocular movements, especially conjugate movements upward.
      (3) Nystagmus.
      (4) Strabismus.
   (b) Central deafness. This is now believed to be due not entirely to pressure on the inferior colliculi but also to involvement of the lateral lemnisci.

3. Cerebellar signs, by transmitted pressure downward against the tentorium.

In addition to the foregoing features, certain other general disturbances of function may be present. These are:

1. Possible endocrine dysfunction in the form of macrogenitosomia praecox. This is a relatively infrequent condition and, when present, has been observed only in males under the age of puberty. Of the 6 patients under the age of fifteen in this series, only one (C.C.) showed this syndrome. In Russell and Sachs'[8] review of the literature there were 17 patients under fifteen years of age with the diagnosis of a pineal tumor. Of this number, 3 presented the clinical picture of macrogenitosomia praecox.

2. Diabetes insipidus. Five of our 16 patients (31 per cent) presented this picture, while it was present in 15 of the 58 cases (26 per cent) reviewed by Russell and Sachs.

3. Dystrophia adiposogenitalis. The symptoms suggested by this descriptive term are infrequently seen in patients with tumors of the pineal body. Inasmuch as a rare case has presented this syndrome, we are including it here for the sake of completeness. In our series there was but one patient (M. J. K.) who was even questionably obese. The remaining patients presented no evidence of a Fröhlich-like syndrome. Marburg[6] believed that the adiposity sometimes seen in patients with pineal tumors was the result of an overfunctioning pineal body. Other investigators have concluded it was due to a hypofunctioning pineal body. Cushing accounted for the adiposity by attributing it to changes in the hypophysis as a result of increased intracranial pressure from the usually marked internal hydrocephalus. The last explanation seems the most logical in view of the fact that it is based on pressure changes, which are known to take place intracranially with these tumors, and is in no way dependent on the questionable hormonal influences of the pineal. That the pineal body is capable of any secretion has been seriously doubted by many investigators, and this is still a debated issue. The experimental investigations of Horrax[3] and others, notably Foà,[2] supported by clinical evidence would seem to suggest that the organ might have some endocrine function up to the time of puberty. If present, this function would appear to be concerned with inhibiting physical and mental maturity. A tumor of the pineal body in a patient who has not yet reached the age of puberty has been assumed to have destroyed this inhibiting influence and to have allowed the precipitation of precocious maturity. The pros and cons of this question were considered at some length by Horrax and Bailey.[4]

Returning now to the specific ocular disturbances which form the main theme of this paper, the incidence and types of these disorders in our 16 patients are presented in Table 1 and they are summarized in Table 2. From these tables it can be seen that a comprehensive examination of the eye is likely to prove highly important in patients suspected of harboring pineal tumors. However, it should be stressed that the routine study of the eye that is included as a part of every thorough neurologic examination is sufficient to discover these signs.

To understand more fully the eye signs in cases of pineal tumor it may be helpful to review briefly the location of some of the structures involved. It can be seen from Fig. 2 how compression of the corpora quadrigemina by a pineal tumor could upset the function of the third and fourth cranial nuclei. The nucleus of the sixth nerve has less chance of suffering any direct damage by a growing pineal tumor, but inasmuch as greatly increased intracranial pressure is a prominent feature in these patients, sixth nerve palsies are frequently encountered (25 per cent of our cases).

Since certain clinical features of pineal tumors concern the pupillary and

TABLE 1

Incidence and types of eye signs in 16 cases of pineal tumor

Patients	Pupils			Diplopia	Nystagmus	Limitation of Upward Gaze	VI Nerve Palsy	Papilledema	Visual Fields
	Appearance	Reaction							
		Light	Accommodation						
1. M.H.	Dilated L>R	Poor	Good	None	None	Upward	Left, occasional	None	Normal
2. V. N.	Normal	Poor	Good	None	Occasional to left	None	Right	5–6 D. bilateral	Normal
3. C. B.	Dilated	Poor	Poor	None	None	Upward	None	? Early	Normal
4. G. B.	Dilated	Poor	Good	None	On lateral gaze	Upward	None	None	Slightly constricted
5. B.S.H.	Normal	Good	Good	On left lateral gaze	On lateral gaze bilat.	None	Slight left	None	Normal
6. M. P.	Dilated	Poor	Poor	None	None	? Upward + Lateral	None	4 D. bilateral	Normal
7. M.J.K.	Normal	Good	Good	None	Slight on lateral gaze	None	None	4-5 D. bilateral	Normal
8. R.M.	Normal	Fair	Good	None	None	None	None	Sec. optic atrophy	Normal
9. E.P.	Dilated	Good	Good	Occasional	On lateral gaze	None	None	4 D. bilateral	Normal
10. C.C.	Normal	Poor	Good	Frequent	On lateral gaze	None	Right	3–4 D. bilateral	Normal
11. J.H.	Normal	Good	Good	None	None	None	None	5 D. bilateral	Left temporal defect
12. B.N.	Normal	Good	Good	None	None	Upward	None	None	Normal
13. B.P.	Normal	Good	Good	Frequent	None	None	None	4-5 D. bilateral	
14. S.F.	Normal	Good	Good	None	None	None	None	None	Bitemporal hemianopsia
15. P.D.	Normal	Absent		None	None	Upward and lateral	None	Bilateral	
16. P.A.P.	Normal	Good	Good	None	None	None	None	None	Normal

oculomotor apparatus, a brief review of the neurologic mechanism involved during normal and impaired function of this apparatus may serve a useful purpose. The nucleus of the third nerve has several parts (Fig. 3). The lateral nuclei are concerned with the function of the extrinsic muscles of the eye, while the Edinger-Westphal nucleus controls accommodation and innervation of the constrictor pupillae. Winkler[9] believes the Edinger-Westphal nucleus has to do with synergic movements of the eye, especially

upward associated movements, in addition to its pupillary action. The afferent arm of the pupillary light reflex is shown in Fig. 4. After the long afferent fibers from the retina reach the pretectal region a synapse occurs and impulses continue to the Edinger-Westphal nucleus, then along the efferent arc to the ciliary ganglion and from there to the constrictor pupillae muscle. Interference with this arc by a lesion anywhere on the afferent side up to the synapse at the third nerve nuclei can cause an Argyll-Robertson pupil, that is, failure of the constrictor action of the pupil to light with preservation of pupillary constriction on accommodation convergence.

While in no instance in this series was there a true Argyll-Robertson pupil, it should be noted that in 50 per cent of the cases there was definite

TABLE 2

Summary of eye signs

Total Cases	Pupils			Diplopia	Nystagmus	Limitation of Upward Gaze	VI Nerve Palsy	Papilledema	Average Diopters Swelling	Abnormal Visual Fields
	Dilated	Impaired Reaction								
		Light	Acc.							
16	5	8	2	4	6	5 Definite 1 (?)	4	9 1 (?)	4	3
	31%	50%	12%	25%	37.5%	31%	25%	56%		19%

impairment of pupillary constriction to light with normal reaction on accommodation. In one patient (P. D.) there was no reaction of the pupils to light. The patient was a three-year-old baby and no mention was made of the reaction of the pupils on accommodation. Pressure of the pineal tumor and edema in the region between the lateral geniculate body and the pretectal area could cause this Argyll-Robertson-like reaction. Interference with the efferent parasympathetic pupilloconstrictor impulses from the Edinger-Westphal nucleus to the ciliary ganglion could explain the presence of dilated pupils. Such dilatation of the pupils was present in 31 per cent of our 16 cases.

Let us now turn to some of the mechanisms that are concerned with control of the extra-ocular movements. The cortical centers that control conjugate movements of the eyes have been identified in the posterior one-third of the second frontal convolution and near the visual center in the occipital lobe. The upward movement of the eyeballs is accomplished by the simultaneous action of the superior recti and inferior oblique muscles, both of which are innervated by branches of the third cranial nerve.

Table 2 summarizes the incidence of the chief eye signs noted in our series of 16 patients with tumors of the pineal body. In 56 per cent of the cases papilledema was noted, with an average of 4 diopters swelling of the

disks. A high degree of increased intracranial pressure is a frequent finding in these patients and is easily explained by the location of the pineal body astride the aqueduct of Sylvius. Any tumor of this organ can blockade the

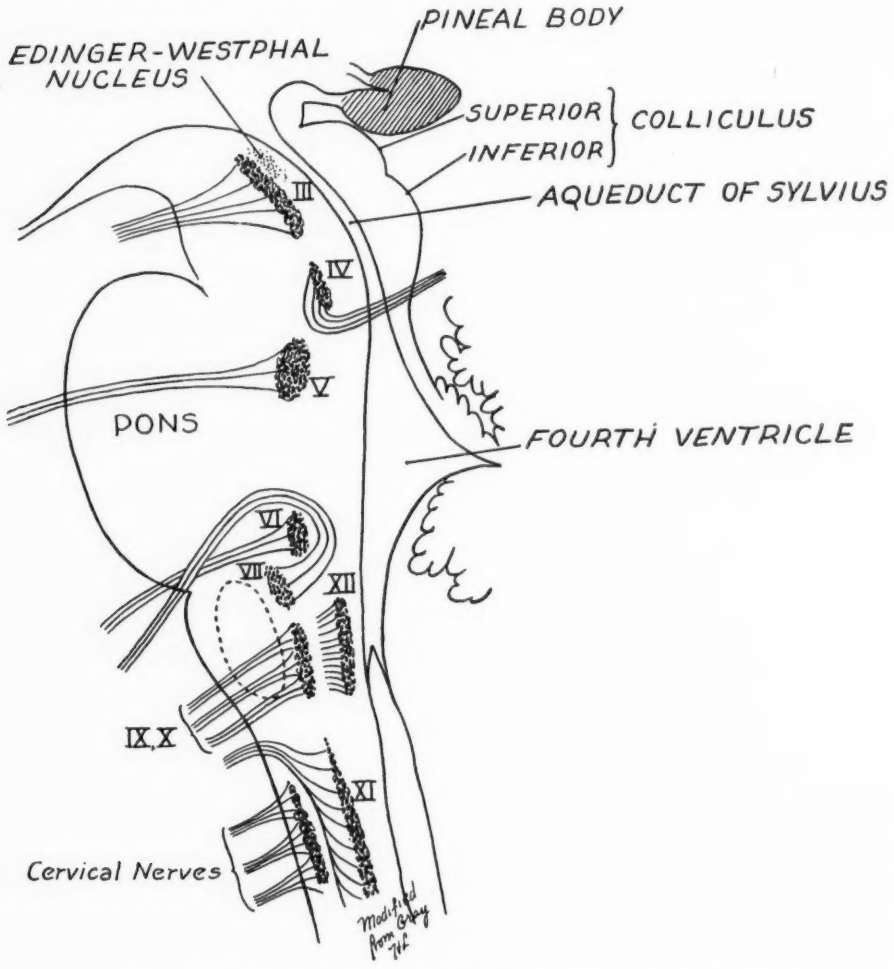

FIG. 2. Position of the cranial nerve nuclei and their relationship to the pineal body.

aqueduct and thereby produce acute internal hydrocephalus. Although 9 of our patients (56 per cent) showed marked papilledema, in 5 of these the swelling of the optic disks and other general complaints associated with increased intracranial pressure were the only definite signs and symptoms to present themselves. A frequent clinical diagnosis in these cases was cerebel-

lar tumor, and in 5 instances exploration of the posterior fossa was carried out, as illustrated by the following case report.

M.J.K. A 25-year-old white, single, female teacher was seen on Jan. 20, 1944, having been referred by Dr. Katherine S. Andrews, of Boston, Massachusetts. The patient complained of unsteadiness in walking, headaches, nausea and vomiting for the past year. Her menstrual periods for the past two years had been definitely irregular and about two years ago she

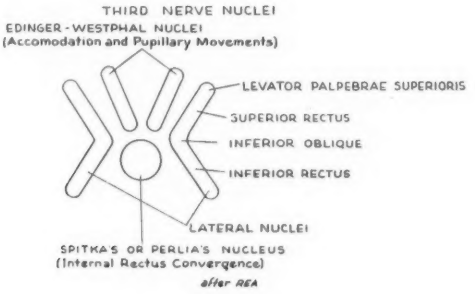

FIG. 3. Detail of third nerve nuclei.

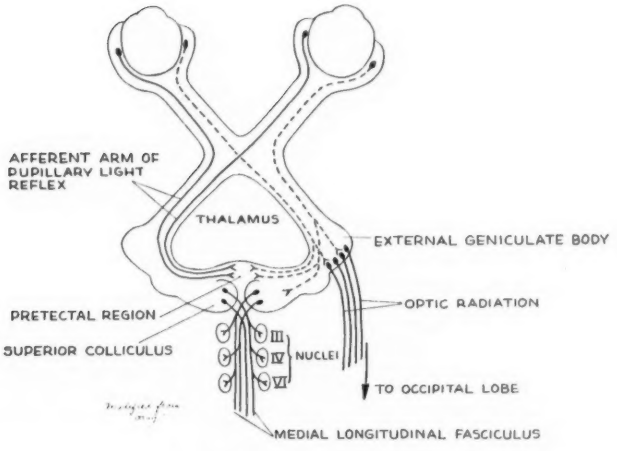

FIG. 4. The afferent arm of the light reflex.

rapidly gained 15 pounds in a few months. During the month before her examination here the patient noted while reading that she would often be unable to see the next word. The history was otherwise negative. She had been seen at another clinic where a diagnosis of probable pituitary adenoma was made.

On examination here, the pupils were normal in size and reacted normally to light and in accommodation. Ocular movements were normal in all spheres. There were a few slow, nystagmoid jerks to the right and to the left. Examination of the fundi revealed 4 to 5 diopters of choking bilaterally, with marked venous engorgement. Romberg's test was markedly posi-

tive, with a tendency to fall to the right. The gait was ataxic, with a list to the right. There was marked hypotonia of the right arm. The remainder of the examination was negative. The clinical impression favored cerebellar neoplasm in the median line, with involvement of the right hemisphere as well. A ventriculogram was performed on Jan. 27, 1944. This revealed a defect in the posterior part of the third ventricle, with marked dilatation of the lateral and anterior third ventricles (Fig. 5).

Because of the marked cerebellar symptomatology and the absence of eye signs it was thought that the shadow in the posterior portion of the third ventricle represented the protrusion of a cerebellar tumor upward rather than a growth in the pineal region. Accordingly,

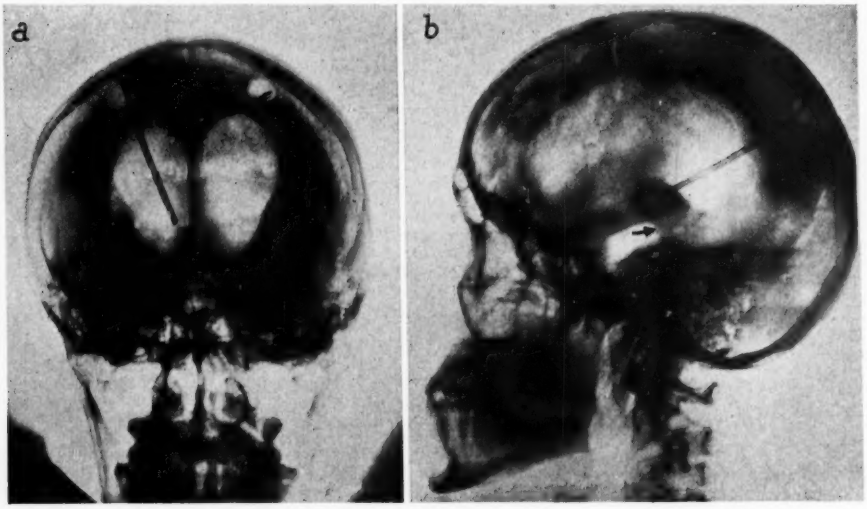

FIG. 5. Ventriculograms of M.J.K. (Case 7). (a) Anteroposterior view: greatly dilated lateral and third ventricles without displacement of third ventricle. (b) Lateral view: marked internal hydrocephalus with defect in the posterior part of the third ventricle (arrow) due to protrusion of tumor. No filling of aqueduct or fourth ventricle.

a posterior fossa exploration was performed, but nothing abnormal was found. Consequently, at the same session, a right occipital bone flap was turned down and the region of the posterior third ventricle was explored after dividing the posterior part of the corpus callosum. A large pineal tumor was disclosed in this location, verified by frozen section, and a grossly complete removal of the tumor was achieved.

After a somewhat stormy convalescence, the patient made an excellent recovery, and has remained very well until the present time, 1½ years after operation.

Comment. This case illustrates how easily a pineal tumor can imitate a cerebellar tumor. It is representative of the 69 per cent of the cases in this series in which extra-ocular movements were well done and without limitation of gaze upward or in any other direction. Parinaud, in 1879, noted the difficulty that patients with lesions in the midbrain, especially near the colliculi, had in upward convergence. Certainly, the finding of paralysis in conjugate movement of the eyeballs above the horizontal is a most signifi-

cant observation and often may be the deciding factor in the localization of a lesion. It should be remembered, however, that many tumors occur in the pineal region without impairment of extra-ocular movements (two-thirds of the patients in our series), and also that tumors in other situations may impair these movements by transmitted pressure.* In their review of 58 verified pineal tumors, 36 per cent of the patients were reported by Russell and Sachs to have had impairment of conjugate movement of the eyes upward. It should also be noted that the pupillary reactions in the case reported above were normal despite the fact that a large tumor was demonstrated in the pineal region. In retrospect, the only clue to the diagnosis of pineal tumor as against cerebellar tumor in this case was the misinterpreted defect in the posterior part of the third ventricle, as shown in the ventriculogram.

DISCUSSION

It is well to note once again that a comprehensive study of the eyes is an especially worthy portion of the general examination in any patient suspected of having a pineal tumor. Conscientious observation of the fundi, the extra-ocular movements and the pupillary reactions in these patients is likely to furnish invaluable information. This, when added to the history, the other physical signs and the ventriculogram, may be of the utmost assistance in formulating the diagnosis.

As has been stated above, the clinical manifestations of pineal tumors may closely resemble those of cerebellar neoplasms. During the study of these patients the differential diagnosis between the relatively frequent cerebellar tumor or the rare pinealoma may be quite uncertain. The cerebellar growth can readily be dealt with directly by exploration of the posterior fossa; the patient with a pineal tumor can usually be offered a better prognosis by subtemporal decompression and radiation therapy. The decision as to the location of the growth, therefore, is not an academic subject. Rather, it is vital to the question as to what type of operation and at what site it is to be undertaken.

The characteristic defect in the posterior part of the third ventricle seen in the ventriculograms of patients with pineal neoplasms must certainly be considered a very significant observation. In a number of cases this finding will prove to be decisive. But there will also be occasions in which even the ventriculogram will not be infallible. The information, however convincing, gained from any diagnostic procedure must be interpreted with due perspective and with due regard to the history and physical findings. A study of the eyes, as an integral part of the general and neurologic examinations in patients in whom a pineal tumor is suspected, can provide useful clues to the correct diagnosis.

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* Not infrequently, patients with acoustic neuromas have considerable limitation of conjugate eye movements upward, and this impairment likewise occurs at times with tumors in the cerebral hemispheres.

SUMMARY

As a result of the study of this series of pineal tumors, the following data may be summarized:

1. The eye signs in 16 cases of pineal tumors are tabulated.

2. Dilated pupils were present in 31 per cent of the cases, and in 50 per cent there was impaired reaction to light. Reaction of the pupils on accommodation was impaired in only 12 per cent of the cases.

3. Papilledema was present in 56 per cent of the cases, with an average of 4 diopters elevation.

4. Upward gaze was limited in 31 per cent of the cases.

5. These tumors often present a clinical picture closely resembling that of cerebellar tumors.

CONCLUSIONS

Tumors of the pineal body can be diagnosed by clinical study and ventriculogram. An accurate diagnosis of these growths may save the patient the risk of an exhausting, perhaps fatal craniotomy undertaken at a different site on the basis of a mistaken diagnosis. By recognizing these neoplasms preoperatively one can offer the patient a less hazardous procedure, usually with an encouraging prognosis, by means of a subtemporal decompression and roentgen therapy. Appreciation of the eye signs produced by tumors of the pineal body will contribute to the earlier diagnosis of these growths.

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INTRACRANIAL ANGIOGRAPHY

I. THE DIAGNOSIS OF VASCULAR LESIONS

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THE SYMPTOMS and signs of vascular lesions within the cranium are not always sufficiently typical to permit accurate diagnosis on the basis of clinical findings alone. Even such aids as spinal fluid examination, roentgenography of the skull and pneumoencephalography may fail to tell the exact story. We now possess in the procedure of cerebral angiography a precise method to demonstrate intracranial blood vessels roentgenographically. Angiography may not only show the site of the suspected vascular lesion but it often reveals its anatomic character as well.

In spite of its accuracy, cerebral angiography should not be indiscriminately employed in suspected vascular lesions of the brain. Firstly, the method requires a painstaking and relatively complicated technique which has been described elsewhere in detail (Moniz,[10] List, Burge and Hodges[9]). Secondly, it must be remembered that concentrated radiopaque material is introduced into a diseased vascular system. Injection of foreign substances under pressure has the potential hazard of increasing vascular damage by producing spasm, thrombosis or even rupture of the vessel, yet, as a rule, no such ill effects need to be feared if proper technique and indications are used. Thorotrast has proven in our hands the most satisfactory contrast medium available and has been used by us without incident, even though it admittedly does not meet ideal specifications. Cerebral angiography should be performed according to the following directives. (1) It is indicated especially whenever surgical treatment is under consideration and a precise diagnosis is required for such therapy. Conversely, the procedure is usually inadvisable when the differential diagnosis is merely of academic value. (2) Angiography is contraindicated in very old patients and those with far advanced arteriosclerosis, hypertension, cardiac decompensation or recent embolic or thrombotic episodes. It is likewise contraindicated in most cases of acute intracranial hemorrhage, yet it is permissible in the exceptional instances in which conservative management carries greater risk than surgical treatment.

Admittedly, angiography has certain technical and diagnostic limitations. It is not possible to inject the entire intracranial vascular system by one single procedure. If both hemispheres are to be studied, bilateral injection is required; carotid or vertebral injection must be used to demonstrate the respective arborizations of these vessels.

TECHNIQUE

Intracranial angiography has been used extensively in the University of Michigan Hospital since 1941. The technique has been previously described in detail[9] and will be briefly reviewed.

Carotid Arteriography. Under local anesthesia the carotid bifurcation is exposed by means of an incision anterior and parallel to the sternocleidomastoid muscle. Segments of the common, internal, and external carotid arteries are dissected free. A rubber strip (split Penrose drain) is placed around the common carotid and another around the beginning of the external carotid. The patient is then transferred to the x-ray table and his head turned to the non-operated side (for the lateral projection). Injection equipment consists of three 10 cc. and three 20 cc. Luer syringes, an 18 or 19 gauge needle of medium bevel, 3–4 cm. in length, and a 10 cm. length of pliable, though non-collapsible rubber tubing fitted on either end with adaptors. This equipment must be tested in advance. The common carotid is elevated without strangulating the vessel, by means of the rubber strip, and the external carotid is pulled upward slightly and purposely strangulated by twisting its rubber strip. After the needle-tube-10cc. syringe combination has been filled with contrast medium, a puncture is made into either the beginning of the internal carotid or the common carotid artery close to the bifurcation. Multiple punctures should be avoided. When the needle is in proper position, the intra-arterial pressure will push back the plunger of the syringe and the needle will be gripped by the elastic arterial wall. Ordinarily three injections are made, two to obtain a stereoscopic pair of arteriograms and venograms in lateral projection and a third for a single arteriogram in anteroposterior projection. Thus a total of five films is exposed. Thorotrast (sterile thorium dioxide in 25 per cent colloidal suspension) is used as contrast medium, each injection consisting of 10 cc. of thorotrast (8–12 cc. may be used). The contrast medium is injected as rapidly as possible and, when the injection is two-thirds complete, the signal for x-ray exposure is given to obtain the arteriogram. Three seconds after the completion of the injection a second exposure is made to record the venogram. Without disturbing needle and tubing, the empty 10 cc. syringe is replaced by a 20 cc. syringe containing Ringer's solution, and rubber tubing and needle flushed to prevent clotting. In the meantime, the x-ray tube has been shifted for stereoscopy. A second injection is then performed in an identical manner. Following the second injection, tubing and needle are again flushed with Ringer's solution, until the head is rotated to the anteroposterior position. The x-ray tube is so angled that its beam will pass through a plane that includes the external auditory meati and a point in the frontal midline, 5 cm. above the glabella. Since this injection requires longer exposure time the signal for x-ray exposure is given immediately after beginning the (third) injection. The strangulation of the external carotid is then released, the needle withdrawn and hemostasis accomplished by firm packing and digital pressure. When all bleeding from the puncture wound has stopped, packs and rubber strips are removed, and the wound is washed with warm Ringer's solution and closed in layers. A pressure bandage (small sandbag) is advisable to prevent after-bleeding.

Vertebral Arteriography. This is most easily performed by retrograde injection of the subclavian artery. The vessel is exposed by transverse supraclavicular incision and section of the anterior scalenus muscle. After occlusion of the peripheral part of the subclavian artery and of the thyreocervical axis, 10–15 cc. of thorotrast are injected into the subclavian artery against the direction of blood flow. X-ray exposure is made at termination of the injection. This method is not absolutely reliable and partial escape of contrast medium into the carotid system may occur.

X-ray Equipment. This consists of a regular stationary skull machine with a line focus, shock-proof tube (focal point 2.3 mm.), energized by a fully rectified transformer operating through a flat radial type of moving Potter-Bucky diaphragm at a target film distance of 90 cm. For lateral projection, an exposure time of 1.5 seconds is used at 20 MA and 75 KV; for anteroposterior projection, exposure time is increased to 3 seconds and kilovoltage to 85.

OBSERVATIONS

Thirty-five cases of vascular intracranial lesions have been studied by this method. Our observations will be divided into the following groups:

(1) Vascular occlusions and coarctation
(2) Intracranial aneurysms
(3) Arteriovenous fistulas
(4) Congenital arteriovenous malformations

1. VASCULAR OCCLUSIONS AND COARCTATION

In massive vascular lesions of the brain, angiography is an excellent method to determine the presence and site of an arterial obstruction. The arterial occlusion occurs more frequently in the carotid artery itself than has been realized (Sorgo[11]). If the thrombosis extends to the customary site of injection, at the level of the carotid bifurcation, angiography is impossible, but in such a case, thrombosis of the vessel is easily recognized during the surgical exposure, as the following observation will show.

Case 1. G. E., 524260, female, age 45. Two months before admission, this patient experienced a right-sided Jacksonian convulsion followed by aphasia. There were two subsequent convulsions of similar character, following which total aphasia and right hemiplegia persisted. Significant findings on clinical examination were total aphasia, spastic hemiplegia on the right, with slight voluntary movements of the leg, weakness of the left radial pulse, and almost complete absence of the left carotid pulse. Angiography was attempted as a means of verifying the tentative diagnosis of left carotid thrombosis.

Surgical exposure of the left carotid at its bifurcation showed both the common and internal carotid arteries to be thrombosed. Sclerotic plaques were visible in the vessel walls. Since no portion of the lumen of the artery was found to be patent, when multiple punctures were made, no injection of opaque material was possible.

If the intracranial portion of the carotid artery is occluded, angiography can be carried out in the usual manner, but the vascular obstruction causes a considerable increase of resistance against the injection of thorotrast. This leads to poor filling of the obliterated vessel and to reflux of most of the contrast material into the circulation of the external carotid artery. As a matter of routine, we strangulate the external carotid artery at the time of injection to obviate this difficulty.

Case 2. A.W.R., 494930, male, age 34. Upon admission, 3 weeks following a stroke, this patient was found to have complete spastic hemiplegia on the right and total aphasia. A provisional diagnosis of extensive vascular brain lesion was made and angiography was employed to provide a more detailed understanding of the suspected disturbance of circulation in the cerebral vessels.

The first attempt resulted in excellent filling of the branches of the external carotid, while very little of the opaque material entered the internal carotid. To prevent selective filling of the external carotid, this vessel was temporarily strangulated and a second injection was made against considerable resistance. Roentgenograms (Fig. 1) showed the internal carotid to be of normal caliber throughout; the ophthalmic artery was even somewhat larger than normal. Just proximal to the origin of the anterior and middle cerebral arteries the vascular lumen was abruptly narrowed to very small caliber. A few very slender and unusually straight twigs of the middle cerebral artery were visible. There was no filling of the anterior cerebral artery.

FIG. 1. Case 2. High-grade occlusion of intracranial portion of carotid artery. Note large ophthalmic artery. Sylvian vessels show minimal filling. There is reflux of thorotrast into external carotid artery.*

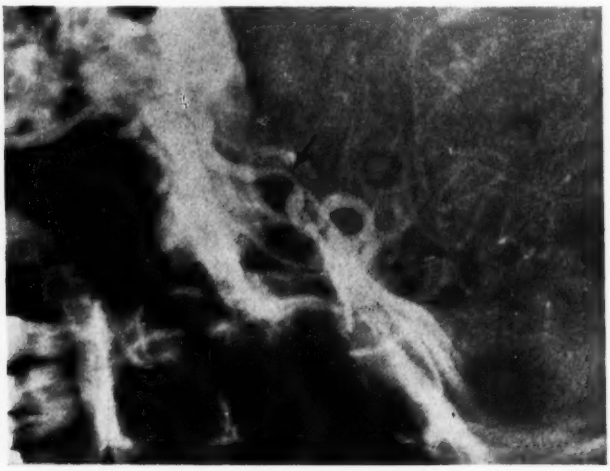

FIG. 2. Case 3. Probable coarctation of internal carotid artery within cavernous sinus.

* Some of the illustrations have been retouched for the sake of clarity of reproduction.

The next unusual case is reported as an example of coarctation of the internal carotid which could be diagnosed only by means of angiography.

Case 3. L.D., 490129, female, age 33. The patient entered the hospital because of right frontotemporal and orbital pain of 3 months' duration, occasionally associated with nausea and vomiting. In recent weeks she had developed diplopia.

When blood pressure and pulse volume were tested, striking differences between the right and left sides were observed. Blood pressure in the right arm was 200/100; in the left arm 158/90. Carotid pulse was faint on the right; powerful on the left. A loud systolic bruit, best heard over the left carotid, somewhat more faintly over the aorta, could not be followed into the skull. The right eye protruded slightly and there was venous stasis in the mildly ptotic upper lid as well as paresis of the superior rectus and the superior and inferior oblique muscles. The right pupil was narrowed. These findings suggested the existence of multiple vascular anomalies, possibly associated with subclinoid aneurysm of the right internal carotid.

By means of angiography the right internal carotid was found to be of abnormally small caliber, no larger than a radial artery, while the cerebral branches were not adequately filled. A second injection, during strangulation of the external carotid, required the use of unusually high pressure. This time coarctation of the internal carotid within the cavernous sinus could be seen in the arteriogram (Fig. 2). Proximal and distal to the narrow point the vessel and its branches were of normal caliber. There was no noticeable alteration in the course of the artery.

At subsequent operation, performed to exclude the possibility of small parasellar tumor, the chiasm and the right parasellar region were exposed. No neoplasm was found. There was no evidence of abnormal bulging of the cavernous sinus, and the internal carotid from its dural entrance to the origin of the cerebral branches appeared normal. One year later, spontaneous disappearance of the presenting symptoms and physical findings had occurred while primary optic atrophy with reduction of visual acuity to 1/60 had developed on the right side. Final diagnosis: Coarctation of the internal carotid, possibly associated with occlusion of the ophthalmic artery.

In chronic encephalopathies, it may be of interest to determine whether the lesion is of inflammatory or of vascular origin. Angiography may answer this problem, as the following case of infantile hemiplegia will show.

Case 4. B.S., 486898, female, age 7. Delivered spontaneously by a mother in eclampsia, this child was cyanotic during the first few days of life. Left hemiparesis and left-sided Jacksonian convulsions had been observed since birth.

Left-sided hemiatrophy and spastic hemiplegia, more marked in the arm, were apparent on initial examination. Pneumoencephalography showed atrophy of the right cerebral hemisphere, particularly of the posterior portion, with definite enlargement of the subarachnoid channels in the Sylvian area. Provisional diagnosis: Encephalopathy of right cerebral hemisphere caused by birth trauma. It was felt that the lesion had resulted from cerebral anoxemia.

Angiography (Fig. 3) showed good filling of the internal carotid and the anterior cerebral arteries on the right, with virtually no filling of the middle cerebral artery or its branches. It seems probable that thrombotic occlusion of the middle cerebral artery was the initial incident which resulted in stunted growth of that part of the hemisphere supplied by this vessel.

2. INTRACRANIAL ANEURYSMS

In spite of many excellent clinical descriptions of cerebral aneurysms (Jefferson,[6,7] Dandy,[5] Krayenbühl[8]), it may be stated that no other diagnostic method surpasses angiography in accuracy. Not only are the location and size of the aneurysm clearly shown, but sometimes multiple aneurysms

are revealed. On the other hand, angiography has its limitations even in this field where it is so specifically indicated. For instance, the aneurysmal sac, as seen in angiograms, may be smaller than the aneurysm found at operation or autopsy, if it is partially obliterated by clot. If the aneurysm or its neck is very small, the pouch may not accept any of the contrast medium; this happened in one of our observations (verified by operation). In other cases, the sac demonstrated by radiopaque medium may appear larger than the true aneurysm, if a rupture has occurred that has been walled off by the neighboring tissue (see below). Within the aneurysm, circulation may be

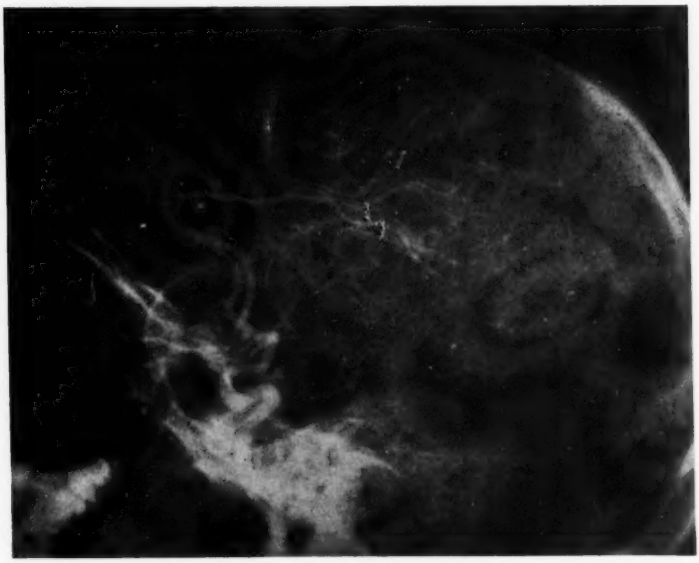

FIG. 3. Case 4. Infantile encephalopathy involving right hemisphere. Both anterior cerebral arteries are unusually well shown, but only a few branches of middle cerebral artery are visible. This suggests old vascular occlusion.

slowed to such an extent that opaque material may remain in the sac throughout both the arterial and the venous phases. Intracranial aneurysms may be extradural or intradural.

(a) *Extradural subclinoid aneurysms* arise from the extradural portion of the internal carotid which passes through the cavernous sinus. The extradural aneurysm rarely bursts, because it is well protected by dura (walls of the cavernous sinus). It acts like a slowly growing tumor in the parasellar region and hence encroaches upon the 3rd, 4th, 5th, and 6th nerves, occasionally also on the optic nerve and chiasm. Sometimes the calcified wall of the aneurysm becomes directly visible on roentgenograms or parasellar erosion, enlargement of the superior orbital fissure and destruction of the

lateral wall of the optic foramen can be demonstrated. Nevertheless, a definite differential diagnosis between aneurysm and parasellar tumor can be made preoperatively only by angiography, as illustrated by the following observations.

Case 5. W.B., 480266, female, age 57. For the past 2 years, the patient had suffered from severe pain on the left side of the face and head, followed by left facial numbness and diplopia.

Examination showed total paralysis of the 6th nerve and pareses of the 3rd and 5th nerves on the left. Roentgenograms of the skull revealed parasellar erosion on the left extending to the floor of the middle fossa and to the tip of the petrous apex. Carotid angiography (Fig. 4) was carried out to establish the differential diagnosis from parasellar neoplasm. It showed a large (plum-sized) subclinoid aneurysm of the internal carotid in the cavernous sinus.

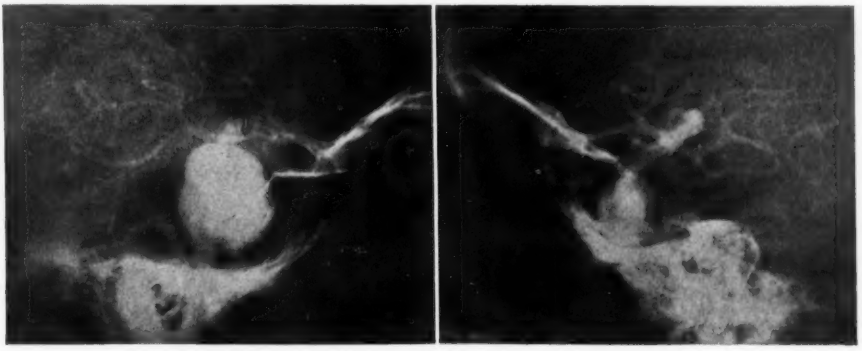

FIG. 4 (*left*). Case 5. Large extradural aneurysm of internal carotid artery.

FIG. 5 (*right*). Case 7. Extradural subclinoid aneurysm of internal carotid artery. Note arteriosclerotic changes of major cerebral arteries.

Case 6. B.H., 472827, female, age 32. This patient suffered from left-sided "migraine" for 4 years, and developed persistent left frontotemporal and orbital pain 4 weeks prior to admission.

Significant findings on admission were: Mild protrusion of left eye with venous congestion of orbital tissues, paralysis of 6th nerve, and hyperesthesia in the first and second divisions of the 5th nerve, on the left side. The left superior orbital fissure appeared to be enlarged on roentgenograms. With the diagnosis in doubt, the parasellar region was explored. Laterally and beneath the eroded left anterior clinoid process, a pulsating extradural mass was found which was verified as aneurysm by aspiration. After temporary improvement, the patient returned 9 months later with a partial 3rd nerve palsy.

Arteriography, then performed, demonstrated the typical saccular aneurysm of the internal carotid in the subclinoid region. Subsequent ligation of the internal carotid at the neck greatly improved the patient's condition.

A third similar observation is described here, mainly because unusually severe arteriosclerosis of cerebral arteries could be demonstrated by angiography in addition to a parasellar aneurysm of the carotid. In this case, angiography initiated an episode of mental confusion and extremely high blood pressure; hence, in retrospect, the procedure must be considered inadvisable in such patients.

Case 7. A.M., 535627, female, age 57. The patient had arterial hypertension with retinop-

athy and aortic insufficiency. Neurologic findings were interpreted as a pseudobulbar syndrome combined with a left parasellar lesion.

Angiography (Fig. 5) confirmed not only the presence of the suspected left subclinoid aneurysm, but showed the major cerebral arteries to be of peculiarly rigid, pipe-like appearance, with changes of caliber.

(b) *Intradural aneurysms* arising from the internal carotid, and from the arteries of the circle of Willis and their branches, have a tendency to rupture before reaching a large size. They are usually congenital in nature in contrast to the common extradural aneurysms of arteriosclerotic origin. The arteriographic appearance of intradural aneurysms is that of a small berry or pear-shaped sacculation with a narrow neck indicating the site of the "blow out" in the artery. As mentioned above, their shadow as outlined by contrast material may appear much larger than the actual size of the aneurysm if a covered perforation is present that is walled off by thickened leptomeninges (false aneurysm). As a rule intradural aneurysms remain clinically silent until they rupture or develop a slight leakage with resulting subarachnoid or intracerebral hemorrhage. A larger clot in the vicinity of the bleeding aneurysm can often be recognized on arteriograms by characteristic displacement of the arteries of the neighborhood.

While spontaneous subarachnoid hemorrhage is frequently caused by a ruptured aneurysm, in not every such case should the patient be subjected to angiography. It appears unwise to perform angiography in the acute stage and the procedure should be limited to quiescent cases with a history of repeated attacks. If it is impossible to localize the aneurysm by clinical means, angiography must be done bilaterally. Seven typical observations may serve as illustrative examples.

Supraclinoid aneurysms, arising at the point where the internal carotid artery emerges from under the anterior clinoid process, extend anteriorly, medially and upward, thus compressing the optic nerve or chiasm.

Case 8. C.R., 524351, male, age 30. After an attack of sneezing the patient developed a violent right fronto-occipital headache, followed by loss of vision of the right eye.

On admission (5 days after onset of symptoms), the patient presented the picture of acute meningeal irritation, with xanthochromic spinal fluid containing 200 crenated erythrocytes per c.mm. The right eye was blind and funduscopic evidence of primary optic atrophy developed after 3 weeks. The clinical diagnosis was subsiding subarachnoid hemorrhage, presumably from an aneurysm of the right anterior cerebral artery.

Angiography (Fig. 6) revealed a pear-shaped aneurysm arising from the supraclinoid segment of the right internal carotid. The aneurysm was in a position to encroach upon the right optic nerve, thus accounting for the blindness. Displacement of the proximal portion of the anterior cerebral artery (upwards, posteriorly and to the opposite side) suggested the presence of a surrounding clot.

The right internal carotid artery was ligated at the neck without incident, and when the patient was last seen, 6 months later, he was symptom free except for right-sided blindness.

Case 9. S.M., 558017, male, age 34. This patient had suffered from repeated attacks of subarachnoid hemorrhage, precipitated by sexual intercourse. Frontal pain, and blurring of vision on the left were the only localizing symptoms. There was a bilateral relative central scotoma for colors.

Angiography demonstrated an unusually large saccular aneurysm of the left internal carotid artery just above the anterior clinoid process (Fig. 7).

At first, the internal carotid artery was ligated in the neck, then, a few days later, the vessel was ligated intracranially, distal to the aneurysm (but below the circle of Willis). At this second operation, the cherry-sized aneurysm was seen to elevate and to compress the left optic nerve and chiasm from below.

The patient tolerated the double ligation very well and his visual disturbances and headaches cleared up.

Aneurysms of the internal carotid artery arising at the origin of the posterior communicating artery tend to develop posteriorly and to encroach upon the oculomotor nerve.

Case 10. M.K., 136218, female, age 35. A week prior to admission, the patient suddenly

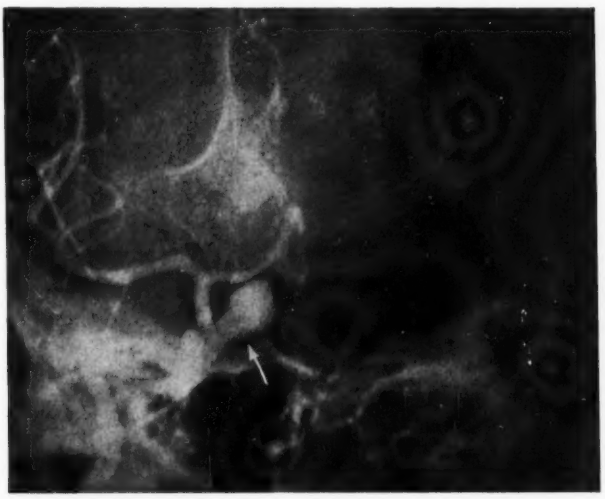

FIG. 6. Case 8. Intradural aneurysm of supraclinoid portion of internal carotid artery. (In frontal projection)

developed severe right-sided headache, stiff neck, nausea and vomiting. She admitted previous syphilitic infection.

She presented signs of meningeal irritation and her spinal fluid was bloody and xanthochromic, with a 4 plus Kahn reaction.

With the diagnosis of subarachnoid hemorrhage on a syphilitic basis, antiluetic treatment was begun. A month later, another attack of subarachnoid hemorrhage occurred, and finally, after forceful vomiting, a complete right 3rd nerve paralysis developed.

The diagnosis of bleeding aneurysm of the right posterior communicating or posterior cerebral artery was then considered most likely.

By means of angiography (Fig. 8), a pear-shaped aneurysm of the internal carotid at the origin of the posterior communicating artery was shown. The aneurysmal sac remained filled also in the venogram, a sign of slowed circulation. Since the lesion appeared to be surgically accessible, a right frontal craniotomy was performed. There was evidence of old subarachnoid hemorrhage and a small aneurysm at the undersurface of the internal carotid.

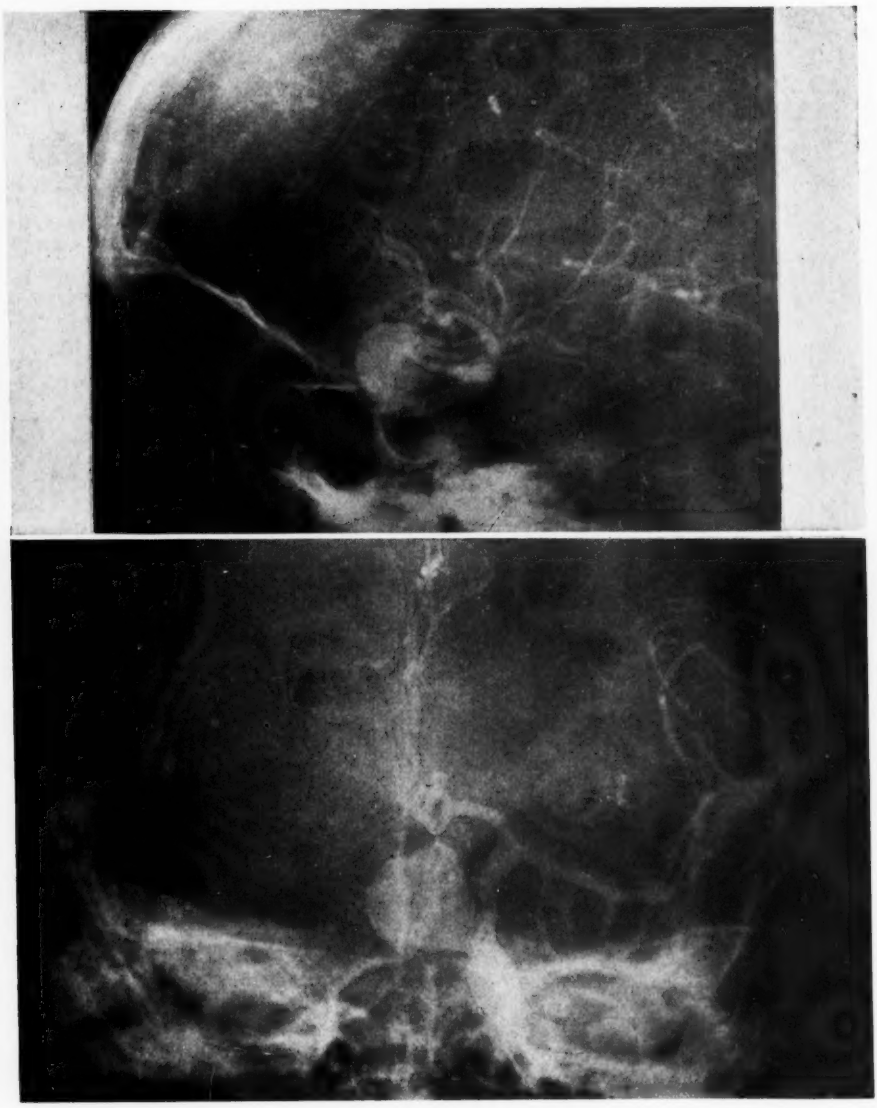

FIG. 7. Case 9. Large intradural aneurysm of supraclinoid portion of internal carotid artery. (*Above*) Lateral view showing also elevation of anterior cerebral artery. (*Below*) Frontal view.

Attempted ligation of the narrow neck of the aneurysm resulted in its rupture and required intracranial ligation of the internal carotid below and above the aneurysm. The patient recovered from the operation with a considerable left hemiparesis, but 7 months afterwards, there were only minimal hemiparetic residuals and the 3rd nerve paralysis had entirely disappeared.

In aneurysms of the middle and anterior cerebral arteries, the clinical picture frequently is so obscured by the signs of subarachnoid and intracerebral hemorrhage that angiography is indispensable for precise diagnosis.

Aneurysm of the left middle cerebral artery with subarachnoid and intracerebral hemorrhage:

Case 11. T.F., 501331, male, age 32. The patient entered the hospital with a 2 days' history of sudden severe left-sided headache, followed by aphasia and confusion.

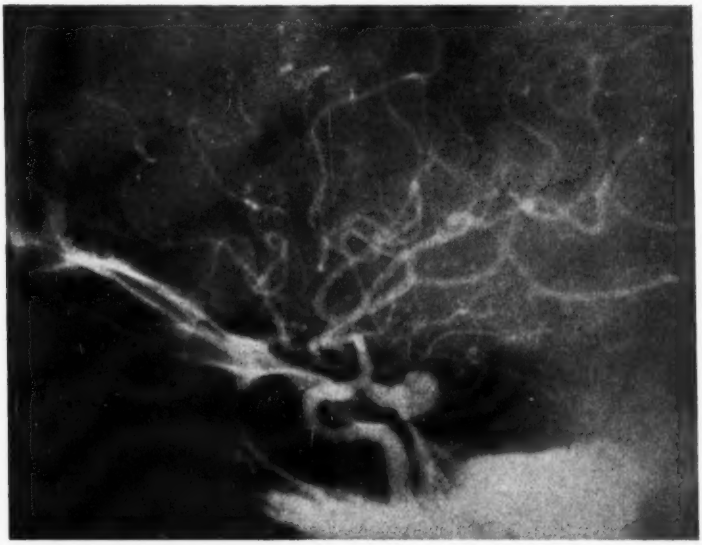

FIG. 8. Case 10. Aneurysm of supraclinoid portion of internal carotid artery arising at origin of posterior communicating artery.

Examination revealed signs of acute subarachnoid hemorrhage and increased intracranial pressure. A mild aphasic and right hemiparetic syndrome suggested the diagnosis of aneurysmal hemorrhage in the left hemisphere. Angiography was performed after the acute clinical signs had somewhat subsided (Fig. 9). A lobulated aneurysm was demonstrated, arising from the left middle cerebral artery within the anterior portion of the Sylvian fissure. Displacement of the anterior cerebral artery to the opposite side indicated hemorrhage into the Sylvian fissure and basal portion of the frontal lobe. Two days after angiography the patient developed right hemiplegia, dilatation of left pupil, became comatose and died.

Autopsy confirmed the angiographic diagnosis. There was a ruptured aneurysm of the left middle cerebral artery. A massive hemorrhage occurred in the temporal lobe, insula and basilar ganglia and perforated into the left lateral ventricle.

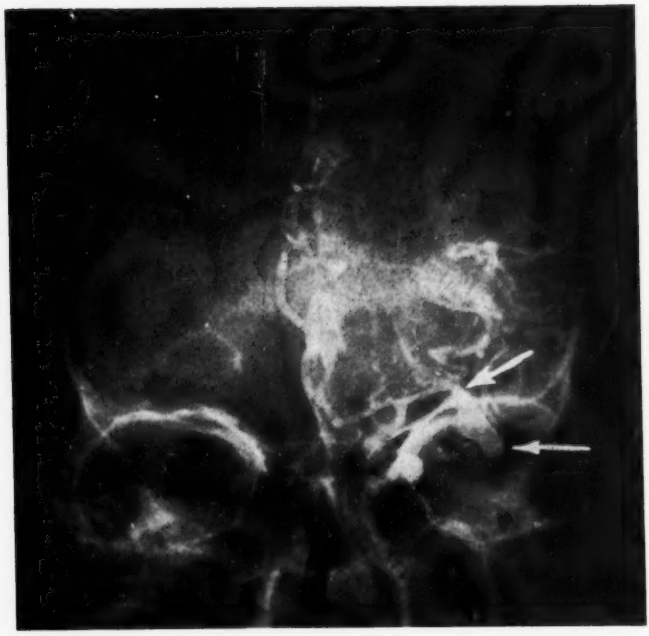

FIG. 9. Case 11. Aneurysm of middle cerebral artery (frontal view); midline shift of anterior cerebral artery indicates intracerebral hemorrhage.

FIG. 10. Case 12. Aneurysm of middle cerebral artery (lateral view); elevation of Sylvian vessels indicates hemorrhage into temporal lobe.

Aneurysm of the left middle cerebral artery with intracerebral and subdural hemorrhage:

Case 12. N.P., 502459, female, age 38. Following a mild head injury, signs of increased intracranial pressure and right hemiparesis developed. Trephination revealed subacute subdural hematoma with evidence of intracerebral hemorrhage. Subsequently the patient became comatose and totally hemiplegic. Reexploration disclosed fresh hemorrhage into the left temporal lobe.

A bleeding aneurysm of the left middle cerebral artery was then suspected and angiography performed after the patient's condition had improved. The arteriogram (Fig. 10) showed not only an aneurysm of the mid-cerebral artery, but also elevation of the Sylvian vessels by hemorrhage into the temporal lobe.

Another observation of double multiple aneurysm may be briefly mentioned here:

Case 13. R.L., 500125, male, age 17. Angiography was performed in this patient because repeated subarachnoid hemorrhages and mild right hemiparesis suggested the presence of a bleeding aneurysm. The arteriogram in lateral projection showed what appeared to be a bilobular aneurysm at the bifurcation of the internal carotid. Operation and subsequent autopsy showed two separate aneurysms, one at the beginning of the anterior cerebral artery and the other at the origin of the middle cerebral artery.

The inaccurate diagnosis in this early case of our series is explained by omitting an angiogram in anteroposterior projection. We now consider this projection essential and more important than a stereoscopic pair of lateral films.

In contrast to the gloomy prognosis of aneurysms of the middle cerebral artery, correct angiographic diagnosis of an aneurysm of the anterior cerebral artery may be rewarded with brilliant surgical results.

Case 14. A.S., 556921, male, age 34. This patient gave a history of three attacks of moderately severe subarachnoid hemorrhage.

The clinical syndrome consisted of signs of meningeal irritation, bilateral papilledema, and pareses of both 6th nerves. Clearcut evidence for localization or even for lateralization was absent. After the subarachnoid hemorrhage had subsided, bilateral carotid angiography was performed. A single lateral arteriogram was obtained on each side, and after it was ascertained that the lesion was on the left, another arteriogram in AP projection was made on that side (Fig. 11). On both projections a pea-sized aneurysm of the left anterior cerebral was clearly visible, but only the AP film accurately showed its position at the proximal portion of the artery 2 mm. from its origin from the carotid artery. By means of a left frontal craniotomy, the region of the anterior cerebral artery was exposed and the aneurysm was trapped by double ligation; several silver clips were placed proximal and distal to the lesion. The aneurysm itself, which was covered by meningeal adhesions and organized clot, was left undisturbed. The patient made an uneventful recovery.

3. ARTERIOVENOUS FISTULA OF THE INTERNAL CAROTID ARTERY IN THE CAVERNOUS SINUS

As a rule, arteriovenous fistula of the internal carotid artery within the cavernous sinus (also called arteriovenous aneurysm) is of traumatic origin and a sequel to basilar skull fracture. Clinically, the presence of pulsating exophthalmos is pathognomonic for these cases. While not indispensable for

diagnosis, angiography gives valuable information regarding the channels of the altered circulation and thereby aids the plan of surgical attack (Wolff and Schmid[12]).

The current of blood in the involved internal carotid is shunted directly into the venous circulation and therefore contributes little to the arterial

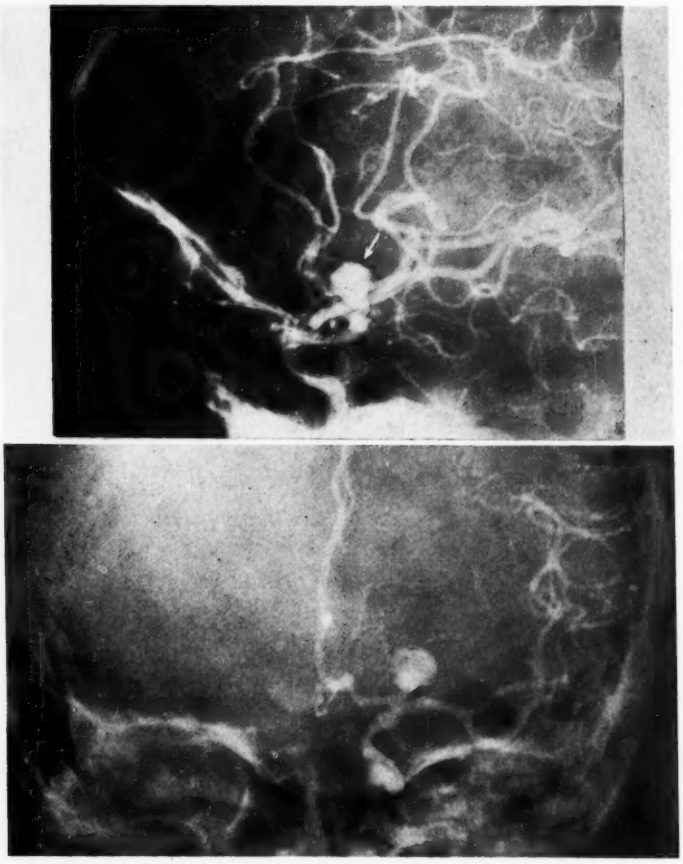

FIG. 11. Case 14. Aneurysm of anterior cerebral artery near origin of vessel. (*Above*) Lateral view. (*Below*) Frontal view showing accurately site of the aneurysm.

irrigation of the ipsilateral cerebral hemisphere. This is clearly borne out by arteriograms in such cases, which show direct passage of the contrast medium from the carotid into the venous system with no filling or only minimal filling of the cerebral arteries. In this respect it is worthy of mention that patients with arteriovenous fistula tolerate the ligation of the carotid very well without showing signs of cerebral anemia (Dandy[3,4]). This is due

to the fact that the cerebral hemisphere on the side of the fistula receives most of its blood supply from the contralateral carotid (via the anterior communicating artery) and from the vertebral system (via the posterior communicating artery). The arterial blood of the carotid mixes with the venous blood of the cavernous sinus and eventually reaches the internal jugular vein by the following pathways (Wolff and Schmid[12]), as shown in Fig. 12.

(1) Cavernous sinus—superior ophthalmic vein—facial veins—internal jugular vein.

(2) Cavernous sinus—inferior ophthalmic vein—pterygoid plexus—facial veins—internal jugular vein.

(3) Cavernous sinus—superior petrosal sinus—sigmoid sinus—internal jugular vein.

(4) Cavernous sinus—Sylvian vein—temporal anastomotic vein of Labbé—transverse sinus—internal jugular vein.

(5) Cavernous sinus—Sylvian vein—Rolandic anastomotic vein of Trolard—superior sagittal sinus—transverse sinus—internal jugular vein.

(6) Cavernous sinus—basal vein of Rosenthal—straight sinus—transverse sinus—jugular vein.

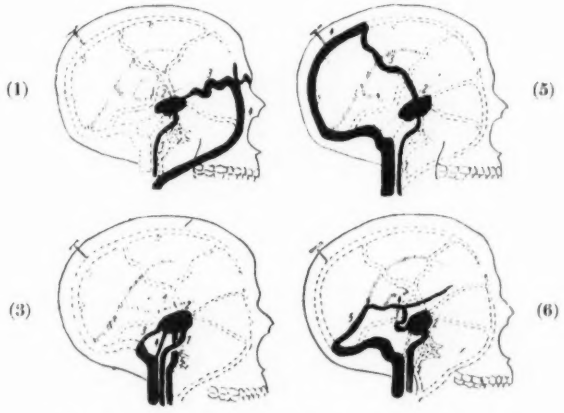

FIG. 12. Schematic drawing (after a paper of Wolff and Schmid[12]) showing venous drainage in cases of arteriovenous fistula in cavernous sinus.

Any one of the above-mentioned venous channels may be well demonstrated at the arterial phase of the angiogram, but it is our impression that usually the superior ophthalmic vein is the most important venous outlet. Since the blood rushes into the cavernous sinus under arterial pressure, all veins and sinuses connected with the cavernous sinus are tremendously dilated. Angiographic demonstration of the main venous return is helpful in planning the surgical treatment of pulsating exophthalmos.

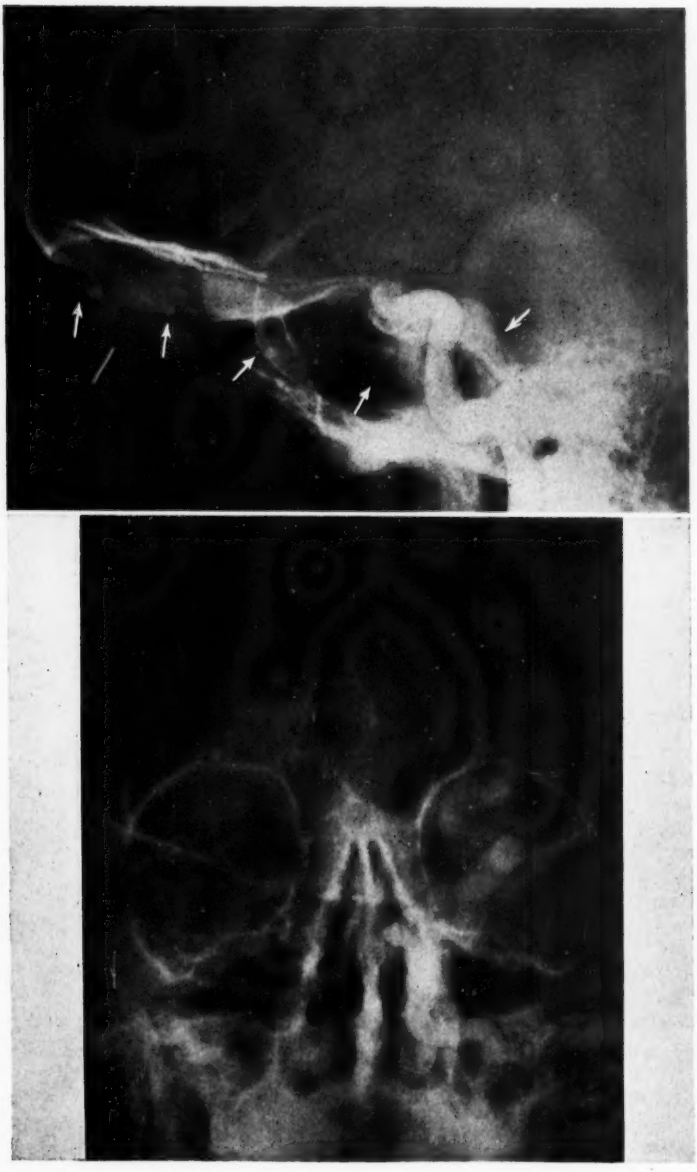

FIG. 13. Case 15. Arteriovenous fistula of carotid artery in cavernous sinus. (*Above*) Lateral arteriogram showing shunting of contrast medium into cavernous sinus, superior petrosal sinus and superior ophthalmic vein (arrows). Cerebral arteries are not filled. (*Below*) Arteriogram in frontal projection shows tremendously dilated superior ophthalmic vein.

The following observations were chosen as typical examples:

Case 15. C.O'C., 487533, male, age 17. Seven years previously, the patient was injured in the left orbital region by a thrown brick. Four months prior to admission, pain and protrusion of the left eye, diplopia, and noises in the head developed gradually. There was a pulsating exophthalmos with considerable venous congestion on the left (21 mm. prominence on left; 13 mm. on right). The left lateral rectus was paralyzed and movements of the other left eye muscles were limited. A loud systolic bruit was audible over left orbit and temple. The left superior orbital fissure was enlarged on roentgenograms. Angiography (Fig. 13) demonstrated the contrast material passing directly from the internal carotid into the cavernous sinus and from there into the tremendously enlarged superior ophthalmic vein. Other venous connections of the cavernous sinus with the basilar and inferior petrosal sinuses were also visible, but the cerebral arteries were not shown. Ligation of the left internal carotid artery at the neck resulted in recession of the exophthalmos to 17 mm. and disappearance of the bruit and diplopia.

Case 16. C.T., 533936, male, age 45. This patient developed a right-sided pulsating exophthalmos following skull fracture. A loud bruit was audible over right orbit and temple. Roentgenograms showed a stellate fracture of the right parietal bone extending to the temporal squama and base of the skull; the right superior orbital fissure was enlarged. Angiography demonstrated direct communication of the internal carotid with the cavernous sinus, but no filling of cerebral arteries. The dilated superior ophthalmic vein, pterygoid plexus, and even the internal jugular vein were visible. Since this patient was not materially improved by ligation of the internal carotid at the neck, further surgical procedures (intracranial carotid ligation and ligation of ophthalmic veins) are contemplated in the future.

4. VASCULAR MALFORMATIONS

Vascular anomalies of the brain have been classified as arterial, capillary, venous and arteriovenous malformations. A detailed description of these conditions can be found in the monographs by Cushing and Bailey,[2] and Bergstrand, Olivecrona, and Tönnis.[1] Except for the capillary anomalies, they can be well demonstrated by angiography.

The most common variety is that of the arteriovenous malformations, also known under the name of cirsoid or racemose angiomas. They consist of a tangle of greatly enlarged arteries forming multiple direct connections with tortuous veins which carry partially arterialized blood. Quite frequently, a conglomeration of arterioles or of vessels of nearly capillary size is interposed between the arteries and veins, thus giving the impression of a true angioma. Arteriovenous malformations most commonly arise from cortical vessels of the cerebral hemisphere; a rarer variant affects the basal vessels and those of the midbrain. Although congenital in character, arteriovenous malformations usually become clinically manifest at middle age, and produce focal convulsive attacks, hemiparesis, and signs of subarachnoid hemorrhage. A bruit may be audible over the lesion and, sometimes, roentgenograms show fine calcifications of the abnormal vessels.

The entire vascular system of the head may be enlarged in these cases and, on surgical exposure, one may find a definite enlargement of the common and internal carotid. The angiograms are pathognomonic and show several huge tortuous arteries leading to a dense tangle of finer vessels from which several veins emerge. The angiomatous formations may receive

collateral blood supply from the opposite hemisphere, especially when situated on the mesial surface of the brain. The angiographic appearance of arteriovenous malformations may have superficial resemblance to angioblastic meningiomas or highly vascular glioblastomas, but these neoplasms always cause considerable displacement of the main cerebral vessels and their blood supply never assumes the huge and bizarre proportions found in arteriovenous malformations.

It should be noted that in arteriovenous anomalies, the cerebral circula-

FIG. 14. Case 17. Arteriovenous malformation of middle cerebral system with cortical angioma in Rolandic area. Note enlarged afferent arteries and huge efferent cortical vein.

tion has a greater velocity than normal because of the enlarged vascular bed and the presence of many wide direct arteriovenous communications. One sees, therefore, simultaneous filling of arteries and veins during the arterial phase of the angiogram. Furthermore, an extremely rapid injection of thorotrast is necessary to obtain a good angiogram, since the contrast medium may be excessively diluted by blood. Three cases may serve as illustrative examples. Arteriovenous malformation of middle cerebral system:

Case 17. W.W., 517519, male, age 30. This patient entered the hospital with the complaints of frontal headaches, right-sided motor Jacksonian attacks and noises in the head. Pertinent findings were: Slight amnestic aphasia, mild hemiparesis involving arm and face, unusual tortuosity of retinal vessels, and a bruit audible over the left carotid artery at the neck.

Angiography confirmed the tentative clinical diagnosis of vascular anomaly of the left frontoparietal cortex (Fig. 14). There was some enlargement and tortuosity of the internal carotid. Several very wide and redundant branches of the middle cerebral artery entered into an angiomatous mass of fine vessels, situated in the Rolandic area. One unusually large vein connected this lesion with the superior sagittal sinus. The simultaneous demonstration of arteries and veins was considered as characteristic of direct arteriovenous connections. Since a direct attack on the lesion in the left motor cortex appeared inadvisable, the common carotid and, later on, the internal carotid were ligated at the neck and deep x-ray therapy was given over the lesion, but no definite improvement was achieved.

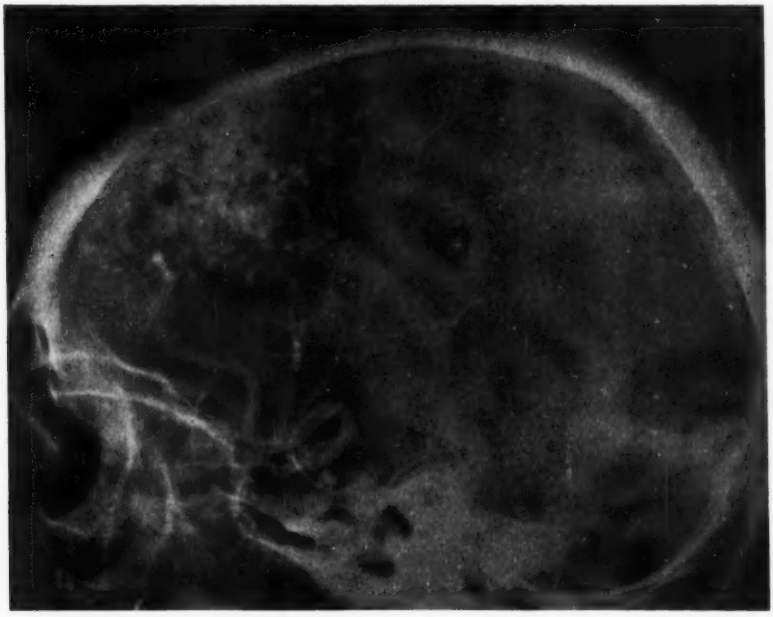

FIG. 15. Case 18. Arteriovenous malformation of anterior cerebral system with cortical angioma in frontal parasagittal area. Note redundant carotid artery and enlarged anterior cerebral vessel.

Arteriovenous malformation of anterior cerebral system:

Case 18. N.L., 527887, male, age 59. For many years the patient suffered from generalized convulsions. Six years prior to admission, right hemiparesis developed which later on had partially receded.

On examination, the patient presented the picture of profound organic dementia with spastic monoparesis of the right leg. Fine calcification was demonstrated roentgenographically in the medial portion of the left frontal lobe. A tentative diagnosis of left frontal oligodendroglioma was made. Angiography (Fig. 15), however, proved that the calcification lay in a tangled network of angiomatous, arteriovenous communications. The arterial supply of the lesion came from greatly enlarged and tortuous branches of the left anterior cerebral artery; the venous drainage flowed into the superior sagittal sinus. The patient was in too poor a condition for surgery and died a year later in a mental hospital. Autopsy confirmed the angiographic diagnosis of arteriovenous malformation arising from the anterior cerebral artery.

Case 19. O.M., 561407, male, age 12. After a questionable head injury 6 days prior to admission, the boy developed severe headaches, followed by convulsions and vomiting. On admission, he presented evidence of recent subarachnoid hemorrhage (bloody xanthochromic spinal fluid), beginning papilledema, and mild transient paresis of the right lower extremity.

After the meningeal irritation had receded, angiography was performed (Fig. 16). It showed a large left anterior cerebral artery feeding an angiomatous conglomeration of small vessels within the basal portion of the frontal lobe. There was a large varix at the posterior part of the angiomatous mass which drained directly into the internal cerebral vein and the vein of Galen. Through a left frontal craniotomy unusually large internal carotid and anterior

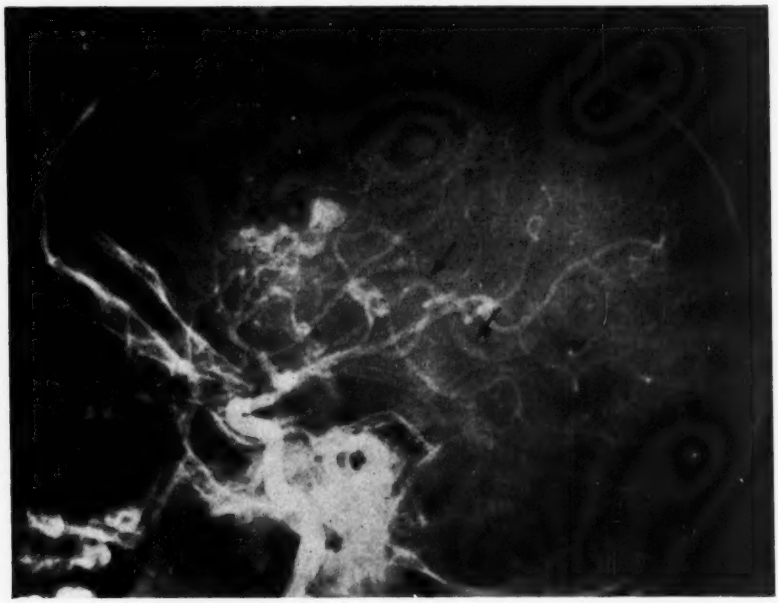

FIG. 16. Case 19. Arteriovenous anomaly of anterior cerebral artery with large venous varix draining into internal cerebral vein and vein of Galen (arrow).

cerebral arteries were exposed. Multiple ligations of the left anterior cerebral and anterior communicating arteries were performed; no other lesion was disclosed. Except for transient paresis of the right foot, the patient made an uneventful recovery.

SUMMARY

Angiography is an accurate method for the diagnosis of vascular intracranial lesions. It may permit one to determine not only the site of the lesion, but often its anatomic character as well. The procedure should be performed only with strict indications. Typical angiographic findings are discussed in the following conditions and supplemented by descriptions of clinical examples: Occlusion and coarctation of cerebral arteries, extradural and intradural intracranial aneurysm, arteriovenous fistula of the internal carotid in the cavernous sinus, and arteriovenous malformation.

Angiography is a valuable aid for planning surgical treatment of certain intracranial vascular lesions.

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HEMATOMAS ASSOCIATED WITH PENETRATING WOUNDS OF THE BRAIN

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PENETRATING wounds of the brain may be considered usefully in three general groups: (1) the gutter type of wound in which depressed bone fragments have lacerated the dura and been driven into the substance of the brain, but no foreign body is present; (2) penetrating wounds in which a metallic foreign body (bullet or high-explosive shell fragment) is retained in the brain; and (3) through-and-through wounds in which a missile has passed completely through a portion of the intracranial chamber. Certain observations have been made in the primary operative management of the latter two types of injury which appear to be of sufficient interest to report at this time. These observations are based on the combined experience of the authors in various forward medical installations (Field and Evacuation Hospitals) in the European Theater of Operations between June, 1944 and March 1945. During this period, 305 penetrating wounds of the brain have been operated upon.*

INTRACRANIAL HEMATOMAS DISTANT TO THE WOUND OF ENTRANCE ASSOCIATED WITH RETAINED FOREIGN BODIES IN THE BRAIN

Neurosurgeons are in general agreement that metallic foreign bodies in the brain should be removed at the time of primary debridement if readily accessible. However, if their removal necessitates an approach through uninjured cerebral tissue, it is felt that the risks of increasing the neurological deficit and of opening new avenues for possible infection from contaminated wounds outweigh the benefits to be gained and such foreign bodies are left in place. The policy has been, then, to remove them subsequently through a clean operative approach only when they are large, very near the surface, or productive of neurological symptoms or signs. But as far as primary operative management is concerned, it must always be remembered, and is strongly emphasized again here, that the pathology created by a missile passing through the brain is much more important than the disposition of the missile itself.

It was not until relatively late in the campaign, November, 1944, that the significance was appreciated of intracranial hemorrhage on the side opposite from the wound of entrance in cases of retained foreign bodies. Since that time, 11 patients have been operated upon who have had sizable

* This series does not include simple compound depressed skull fractures but only those with laceration of the dura and actual brain damage.

hematomas located either in the opposite subdural space or in the substance of the brain in or adjacent to the tract made by a metallic foreign body at a site distant to the point of penetration. In retrospect we feel sure that similar lesions were overlooked in earlier cases.

If a foreign body, particularly a small one, is visualized on x-ray examination close to the cortex of the side opposite that of entrance, there is no way of knowing whether its location marks the end of the tract through the brain or whether it reached the meninges or skull and bounced back into the brain without fracturing the bone. If such a missile did reach the surface, the possibility of its tearing cortical vessels and causing hemorrhage into the subdural or subarachnoid spaces or into the substance of the brain itself is not unlikely. In fact, hematoma in all three locations may be present simultaneously since these spaces may be connected at the instant of trauma. Also, it seems probable that cortical vessels, or more likely some of the vessels leading from the cortex to the dura in the region of the dural sinuses, may be ruptured without the foreign body ever actually reaching the surface, as large subdural hematomas have been encountered where it was impossible to find any evidence of a missile tract reaching the surface (Cases 1 and 2).

When there is intracranial hemorrhage at a point distant to the wound of entrance, this blood does not escape and a space-occupying hematoma is formed. As a result, the decompression automatically obtained in the greater percentage of penetrating brain wounds is not present and the pathological physiology assumes the character of that seen in closed head injuries, where in addition to actual cerebral damage at the moment of trauma, subsequent injury due to cerebral compression may be a more important factor.

In the absence of direct injury to vital centers of the brain stem, it is apt to be this cerebral compression from intracranial hemorrhage that is the lethal factor in penetrating wounds of the brain. If intracranial hemorrhage is massive and diffuse throughout the brain, or if it fills the ventricular system with clots, the result may be fatal before the patient reaches the hospital. If such a patient does reach the hospital, surgical procedures so far have proved of little avail. However, if the hemorrhage is limited to the extradural or subdural spaces or to any one area of the cerebrum so that a localized subcortical hematoma, even though large and even though extending into one of the lateral ventricles, is formed, then surgical evacuation is often possible and an early attack may be life-saving.

It has become our policy, therefore, when a foreign body has crossed the midline and lies within 2–3 cm. of the cortex of the opposite side by x-ray, to place a burr hole on this side as nearly as possible over the location of the missile. This is done whether or not a fracture on this side is visualized by x-ray. If no extradural bleeding is encountered, an opening is made in the dura. If no subdural hematoma is present and the cortex appears entirely normal, nothing more may be done. However, if the cortex is soft-

ened or contused, a brain cannula is directed toward the location of the foreign body. It is easy to tell the difference between a wound tract and normal brain if the cannula is manipulated gently.

If the foreign body has reached the surface and bounced back, as apparently occasionally happens, some hematoma formation is almost certain. It may be possible to retrieve the foreign body itself and this should be attempted if it is of any size or is readily located (Cases 3, 4, and 5). However, the evacuation of any hematoma and macerated tissue present in the neighborhood of the foreign body or its pathway through the brain is felt to be much more important. The tract is followed right down into the ventricle if necessary to accomplish a thorough debridement (Case 6). The burr hole is enlarged sufficiently to carry this out with safety.

Such an exploratory burr hole is easily made under local anesthesia either before (Cases 3 and 4) or after (Cases 1 and 2) debridement of the wound of entrance and adds very little to the length or shock of the operative procedure. In cases in which the clinical picture of cerebral compression is out of proportion to the extent of the visible brain wound, this exploration for a distant hematoma is especially recommended before attacking the wound of entrance.

Brief reports of cases illustrating this type of injury are presented.

Case 1. Admitted to an Evacuation Hospital on Jan. 14, 1945 in very poor condition. He was in moderate shock, deeply comatose, with stertorous respirations. There was slight improvement following oxygen, suction of nasal and oral secretions, and shock therapy. There was a 2.0×2.0 cm. wound, just posterior to the lateral margin of the left orbit, with herniation of brain tissue. The left eye was proptosed onto the cheek. There was left-sided motor weakness and intermittent twitching of the left side of the face and neck. X-rays revealed a depressed fracture of the left frontal bone with fragments driven into the frontal lobe. A metallic foreign body lay in the midparietal region on the right about 3 cm. from the surface.

Under local anesthesia the left frontal wound was debrided through an eyebrow and temporal incision. An estimated 30–40 cc. of macerated brain and numerous indriven bone fragments were removed. The dural defect was sutured and the scalp wounds closed tightly. The eye was enucleated. A parietal burr hole was then placed on the right over the estimated site of the foreign body, revealing an extremely tense blue dura. The burr hole was enlarged and 80–100 cc. of dark fluid and clotted hematoma were evacuated from the subdural space. No brain wound was visible.

The postoperative course was very stormy for 4 to 5 days with fever, irregular breathing, and markedly bloody spinal fluid under greatly increased pressure. He was treated with oxygen, frequent suctioning, bi-daily lumbar punctures, intrathecal (10,000 units daily) and intramuscular (20,000 units every 3 hours) penicillin, and parenteral fluids. Twitching stopped on the 1st postoperative day. Wounds healed primarily and the patient was taking fluids and beginning to talk at the time of evacuation on the 8th postoperative day. His left hemiparesis was improving.

Case 2. Admitted to an Evacuation Hospital on Jan. 17, 1945 in poor condition. He was comatose and in moderate shock. There was a 2.5×2.0 cm. wound in the left frontal region with herniation of brain tissue. Left-sided weakness was noted. X-rays revealed a depressed fracture in the left frontal region with bone fragments driven well into the frontal lobe. A metallic foreign body was seen in the right midparietal region, about 2.5 cm. from the surface.

Under local anesthesia, the left frontal wound was debrided. An estimated half of the left

frontal lobe was macerated and was removed with gentle suction. Numerous indriven bone fragments were removed, some of which had lacerated the frontal horn of the left lateral ventricle. It was possible to pass a lighted retractor through the defect in the falx and debride the tract well into the opposite hemisphere. The dural defect was repaired with a free graft of pericranium and the scalp closed tightly. A parietal burr hole was then placed on the right side over the estimated site of the foreign body. The dura was distended and blue, and on opening it more than 60 cc. of dark subdural clot were evacuated.

Immediately following operation the patient's condition was much improved. He continued to improve steadily and was evacuated afebrile, conscious and taking fluids well by mouth on the 6th postoperative day. Wounds healed primarily.

Case 3. Admitted to an Evacuation Hospital on Dec. 20, 1944 in profound shock. He had lost considerable blood from extensive leg wounds and was unconscious. There was a 2.0×3.0 cm. wound in the right occipitotemporal region through which a 1.0×0.5 cm. metallic foreign body had passed posteriorly to lodge beneath the bone in the left occipital region. X-rays showed many intracerebral bone fragments in the right occipitotemporal region but no fracture on the left. Intensive shock therapy was given with response.

Under local anesthesia a small burr hole was first placed over the location of the foreign body and a silver-dollar-sized area of bone rongeured. The dura was discolored and upon incising it about 30–40 cc. of old clot were evacuated. A ventricular needle was introduced, localizing the foreign body, and this was removed. It was about 0.5 cm. beneath the cortex and surrounded by 10 cc. of clotted blood. The tract was then irrigated almost to the midline. The patient's condition, especially his irregular pulse and difficult respiration, showed some improvement and a routine debridement of the right occipitotemporal wound of entrance could then be accomplished.

The patient was evacuated on the 7th postoperative day, with a left homonymous hemianopia but otherwise in excellent condition.

Case 4. Admitted to an Evacuation Hospital on Jan. 3, 1945. He was stuporous, with a blood pressure of 180/50 and a pulse of 48. There was a 10.0×2.0 cm. wound in the left frontotemporal region with brain herniation. Right hemiparesis was present. X-rays showed numerous intracerebral bone fragments in the left temporal area and two pea-sized metallic foreign bodies close to the midline of the left occipital region and lying about 2 cm. beneath the bone.

Under local anesthesia a burr hole was first made over the expected site of the fragments and the dura quickly opened. A minimal amount of subdural blood was present but the brain was bulging. On inserting a ventricular needle, considerable subcortical hematoma was encountered. Roughly 60 cc. of old clot were evacuated and the foreign bodies removed. Routine debridement with temporal fascia repair of the dural defect at the entrance wound was then accomplished.

The patient was evacuated on the 8th postoperative day with a right hemiparesis and right visual field defect. He was fully conscious, eating well, and had well healed wounds.

Case 5. Admitted to an Evacuation Hospital on March 9, 1945 in fairly good condition. He was conscious but semi-stuporous and had weakness of the right leg. There was a 1.0×3.0 cm. wound of entrance in the right inframalar region. X-rays revealed a metallic foreign body (machine-gun bullet) just to the left of the midline and just beneath the calvarium in the parietal area. They also showed a fracture of the floor of the frontal fossa on the left.

Under local anesthesia a transverse incision was made over the vertex and a burr hole placed over the site of the foreign body. The dura was deep blue and tense, and on opening it 50–60 cc. of dark liquid and semi-solid subdural hematoma were evacuated. The bullet was found lying in the superficial cortex, with its point lying against the intact longitudinal sinus. No tract through the brain could be demonstrated and reviewing the X-rays it was assumed that the bullet had penetrated the floor of the left frontal fossa after crossing from the right

and then scooted around beneath the dura to the vertex. Operative and entrance wounds were closed tight.

Postoperative course was uneventful. The patient was out of bed and afebrile when evacuated on the 4th postoperative day. He still had weakness of the right foot.

Case 6. Admitted to an Evacuation Hospital on March 1, 1945 in very poor condition. He was in moderate shock and in deep coma with slow respirations. Reflexes were all markedly depressed and the extremities flaccid. The right pupil was dilated and fixed; the left was dilated but reacted to light. There was a 2.0 × 3.0 cm. wound in the right frontal region with herniation of brain tissue. X-rays revealed a right frontal depressed fracture with indriven fragments of bone, a small metallic foreign body in the midline and a larger foreign body about 1 cm. from the surface low in the left postparietal region.

Under local anesthesia the right frontal wound was debrided. An estimated 50 cc. of macerated brain tissue and numerous indriven bone fragments were removed from a tract that entered the frontal horn of the lateral ventricle. It was possible to lift considerable clot from the lateral ventricle and also through the foramen of Monro from the 3rd ventricle. The dural defect was repaired with a pericranial graft and the scalp closed. A burr hole was then placed over the site of the foreign body on the left. No subdural hematoma was present. The cortex was contused and a ventricular needle dropped easily into a tract from which an estimated 15–20 cc. of dark fluid and clotted blood were evacuated. The tract was debrided down to the ventricle on this side, which was irrigated gently of old blood-stained fluid.

After operation the patient's condition was immediately improved and he continued to improve slowly during the next 4 days. At this time it was necessary for the operator to move elsewhere and the further course cannot be given at this time.

HEMATOMAS ASSOCIATED WITH THROUGH-AND-THROUGH WOUNDS OF THE BRAIN

General surgeons have always recognized that when a high-velocity missile passes through any part of the body, there is greater damage where it emerges from the tissues than where it enters. It has also long been appreciated by neurosurgeons that in through-and-through wounds of the brain due to such missiles the wound of exit is larger than the wound of entrance. In our experience of 50 operated and many other fatal unoperated through-and-through brain wounds, this observation has certainly been confirmed. At the exit wound there is always more destruction of soft tissues and more extensive comminution of the skull.

Recently, we have also noted, as a corollary observation resulting from our interest in distant hematomas, that when a foreign body passes completely through a portion of the intracranial chamber there is very apt to be extensive hemorrhage at the wound of exit. This is logical because of the large number of cortical vessels which are usually torn. Hematomas, therefore, which may be massive and which may be extradural, subdural, subcortical, and may even extend into the lateral ventricle (Case 7), depending upon the peculiarities of the individual lesion, are often associated with the exit wound. We are not referring here to the ordinary pulpification of brain and small hemorrhages which occur all along the course of every brain wound, but rather to really sizable hematomas usually located at or near the exit of the tract from the brain.

The significance of such a hematoma in the surgical management of

these patients has become more and more apparent. Since appreciating its importance we have adopted the policy of always exploring the wound of exit first in through-and-through brain lesions if the two wounds are to be approached separately. Evacuation of a hematoma in the exit wound as quickly as possible may result in an improvement in the patient's general condition, which is usually precarious in this type of injury. It may be possible then to go ahead and perform a thorough debridement and primary repair of the dura and scalp in a patient whose condition otherwise would be too grave to permit this much surgery (Cases 7 and 8). If necessary, treatment of the wound of entry may even be delayed for 24 hours or more after this preliminary debridement of the exit wound and supportive care given until the patient's general condition warrants going ahead with a definitive surgical procedure. Penicillin and sulfonamide chemotherapy have made such a delay possible without significant added danger of infection.

It is now routine with us, therefore, if the entrance and exit wounds are sufficiently distant to indicate separate operative approaches, always to attack the wound of exit first and proceed according to the findings there and the patient's general condition. Whether or not a hematoma is found associated with the exit wound, debridement of this wound first has proven worth while and should increase the chances of recovery in some of these critical injuries.

Selected cases of this type of lesion are presented for illustration.

Case 7. Admitted to an Evacuation Hospital Dec. 12, 1944 in very poor condition. He had a right hemiplegia and exhibited difficult stertorous respirations. The right occipital area showed herniation of brain from a 0.5 cm. entrance wound. There was also herniation of brain from a 2.5 cm. exit wound in the left temporal area. X-rays showed a 2.0 cm. oval bony defect at the entrance wound and an irregular 3.0×2.5 cm. bony defect at the exit wound with several bony fragments extruded with the herniating brain.

Under local anesthesia, the left temporal wound of exit was operated upon first. Fully 50 cc. of intracerebral clot extending down to the ventricle were evacuated. The ventricle was then irrigated and several stringy clots removed. The patient's improvement after this procedure was striking. His respirations and pulse immediately improved and the right occipital wound of entrance could then be routinely debrided and repaired.

He was discharged on the 7th postoperative day, showing no improvement in his right-sided paralysis. Both wounds were well healed and he was eating well.

Case 8. Admitted to a Field Hospital on Jan. 4, 1945 in extremely poor condition. He was semi-conscious, irrational, and in moderate shock with irregular respirations. A metallic foreign body could be felt beneath the skin just above the right ear. The left eye was collapsed and the medial border of the orbit lacerated. X-rays revealed fracture of the left frontal, nasal and ethmoid bones and extensive severely comminuted fracture of the right temporal bone. A metallic foreign body lay in the soft tissues above the right ear.

Under local anesthesia a temporal incision was made on the right, and the foreign body and comminuted fragments of the temporal bone removed. An estimated 30 cc. of macerated brain from the temporal lobe and at least another 30–40 cc. of dark clot were removed from the exit of the brain wound. The dural defect was repaired using a pericranial graft and the scalp closed tight. Following this the patient's condition was definitely improved. The left eye was then enucleated, the orbit debrided, including comminuted fragments of the frontal and ethmoid bones, 15–20 cc. of macerated brain debrided from the entrance of the brain

wound, and the dural tear repaired. This wound was closed by a 2-layer approximation of orbital structures and tight closure of the conjunctival margins.

Postoperatively, he improved very rapidly and at 5 days was alert, afebrile and taking adequate nourishment by mouth.

Case 9. Admitted to an Evacuation Hospital on March 28, 1945 in very poor condition. He was in moderate shock and profound coma, with no response to painful stimuli. There was a left facial paralysis and a left hemiplegia. Respirations were Cheyne-Stokes and oral secretions drained from the mouth. The right pupil was larger than the left. There was a 1.0 cm. wound of entrance low in the right occipital region with herniation of brain, and a 2.0×2.0 cm. wound of exit in the right frontal region at the hair-line, also with herniation of brain. X-rays revealed defects in the skull corresponding to these scalp wounds and no foreign body.

Under local anesthesia the wound of exit was first excised. On removal of comminuted fragments of the frontal bone, 15–20 cc. of extradural clot were found. When the brain wound was uncovered, at least 50 cc. of clotted dark blood emerged under pressure from the brain tract. After debridement of the tract, the dural defect was repaired with a pericranial graft. It was noted at once that the patient's breathing became more regular and his pulse of a much better quality. The wound of entrance in the occipital region was then debrided in the routine manner.

Postoperatively there was steady improvement. He was afebrile after the 3rd day. He began to move his right side and respond to pain on the 1st postoperative day and by the 4th day he took fluids by mouth and responded accurately to questions. His left hemiplegia remained complete. Wounds healed primarily.

Case 10. Admitted to an Evacuation Hospital on Dec. 3, 1944. He was irrational and in extreme shock. In addition to a severe perforating head wound, with entrance in the right frontal and exit in the left frontal regions, he had extensive subcutaneous emphysema from a chest wound with broncho-pleural involvement. The chest condition was controlled with thoracentesis and insertion of a flutter valve. X-rays showed extensive bony destruction in the right and left frontal areas.

Under combined local and intravenous pentothal anesthesia, the exit wound was attacked first. Debridement here revealed a considerable amount of pulped brain and dark clot which was removed. Fully one half of the left frontal lobe was involved. The right side was then operated upon and it was found that about one quarter of the right frontal lobe was involved. Routine tight closure of both was then accomplished.

Patient did well postoperatively and left the hospital on the 14th day with both wounds fully healed and his chest condition controlled.

Case 11. Admitted to an Evacuation Hospital on Dec. 16, 1944. He was semi-stuporous but moved all his extremities. The reflexes on the left were hyperactive and a left Babinski response was present. Breathing was regular and his condition in general was good. He had a 1 cm. wound of entrance in the left parietal region and a 4 cm. right temporoparietal exit wound.

Under local anesthesia the exit wound was first operated upon and no gross hematoma was found. The left side was then debrided and closed in the routine fashion. It was evident that considerably more destruction of brain tissue was present on the right side than on the left. Both dural defects were closed with fascial transplants.

The patient was discharged on the 6th postoperative day in good condition after an uneventful postoperative course.

This case is included to illustrate a simple through-and-through wound of the brain without hematoma formation either in the ventricular system or at the wound of exit, and to point out how much better the general condition

of such a patient is apt to be than when a clot is present. Nevertheless, the more extensive damage at the exit wound is again noted, and the importance of debriding this wound first again emphasized.

CONCLUSIONS

1. Subdural hematomas have been found 7 times and subcortical hematomas 4 times on the side opposite the wound of entrance in penetrating wounds of the brain, when the foreign body was seen by x-ray to have crossed the midline and lie near the opposite cortex.

2. It is recommended, therefore, that whenever a missile traverses a portion of the intracranial cavity and lodges within 2–3 cm. of the surface at some distant point, an exploratory burr hole be placed over this point. If no subdural hematoma is present, a brain cannula should be inserted to explore for a subcortical hematoma in the neighborhood of the foreign body or the tract made by its passage.

3. Such a procedure is carried out easily under local anesthesia in a few minutes' extra operating time. Since its importance was appreciated, results have been sufficiently gratifying to warrant this report for general consideration.

4. In through-and-through wounds of the brain, the wound of exit always exhibits greater damage than the wound of entrance and frequently is the site of a large hematoma.

5. It is recommended, therefore, that when entrance and exit wounds are situated so as to indicate separate operative approaches, the exit wound always be handled first, since the early evacuation of a hematoma or collection of macerated brain tissue here will usually result in immediate improvement of the patient's general condition and may occasionally relieve an otherwise fatal issue.

6. Illustrative cases of these two types of injury have been selected and are presented briefly.

Note: Between March, 1945, when this report was prepared, and May, 1945, when the European campaign ended, several additional similar cases of hematomas distant to the wound of entrance were observed by the authors or reported to them by other neurosurgeons.

THE INCIDENCE OF INTERHEMISPHERIC EXTENSION OF GLIOBLASTOMA MULTIFORME THROUGH THE CORPUS CALLOSUM

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MODERN ADVANCES in neurological surgery permit the surgeon to successfully remove enormous amounts of brain tissue. As a result, the patients with malignant brain tumors have oftentimes been subjected to extensive resections, lobectomies, and even hemispherectomies, in the hope that a complete excision of the gross tumor and adjacent microscopically invaded tissue would effect a cure. Such operations for glioblastoma multiforme are almost always ultimate failures (disregarding the immediate surgical mortality) because a high percentage of glioblastomas extend into the opposite hemisphere through the corpus callosum, and there is therefore recurrence.

At the suggestion of Dr. Eric Oldberg, in order properly to evaluate this disheartening prospect, the recent postmortem cases from this clinic have been collected and reviewed. This study disclosed that in 28 consecutive cases of glioblastoma multiforme of the cerebral hemispheres (pathological diagnoses by Dr. Percival Bailey) autopsied since January, 1941, 21 (75 per cent) had definite gross interhemispheric extension through the corpus callosum, visible to the naked eye.

Of the 28, the main tumor mass was found to be in the right hemisphere in 14 cases, in the left hemisphere in 11 and to be centrally located in the corpus callosum in 3 cases. Of the remaining 7, in 2 cases (1 parietal and 1 temporal) there was questionable extension, and in 5 cases (1 frontal, 3 temporal and 1 parieto-occipital) there was no gross callosal invasion. Sections of the corpus callosum in these seven cases were not examined microscopically, however, since this study is concerned only with extensions that would be visible to the surgeon, were he able to explore the corpus callosum.

The location of the main tumor mass in the 21 cases that showed gross interhemispheric connections was divided as follows: 8 frontal, 7 parietal, 2 central, 1 occipital and 3 callosal. The age, sex, duration of history and findings in all cases were characteristic of cerebral glioblastoma multiforme. The occurrence and extent of involvement of the opposite hemisphere could rarely be anticipated antemortem from clinical findings except in the three cases of gliomas of the corpus callosum. These were readily noted on ventriculography. Figs. 1, 2 and 3 demonstrate typical examples of involvement in the anterior, middle and posterior portions of the corpus callosum.

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The gross directional manner of extension of glioblastomas is influenced by the course of large fiber pathways in the brain.[1,3] Thus, the tumors arising

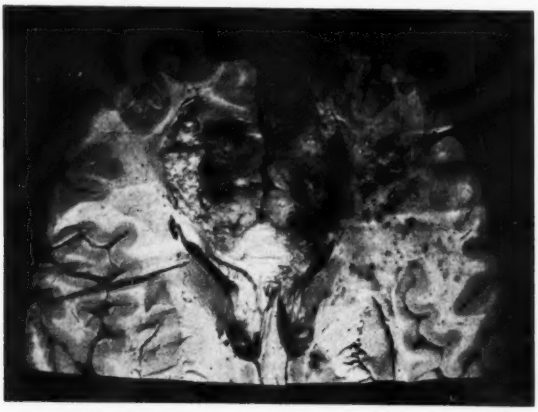

FIG. 1. A large infiltrative right frontal glioblastoma which has crossed through the rostral portion of the corpus callosum to involve the left frontal lobe.

FIG. 2. Bilateral extension into the hemispheres by a glioblastoma of the body of the corpus callosum.

lateral to, or above, the corpus callosum are more often found growing into it parallel to the fibers and thus crossing to the opposite side, the corpus callosum being a broad commissure between the frontal, parietal and oc-

cipital lobes. Tumors arising in the regions below the corpus callosum are largely confined below it, and tend to invade the basal structures, such as the thalamus and peduncles along the corticospinal pathways, rather than to cross callosally to the opposite hemisphere.

H. J. Scherer[2] in 1940 discussed the microscopic characteristics of glioma growth. The incidence of microscopic evidence of extension into the opposite hemisphere is somewhat lower than in our series.

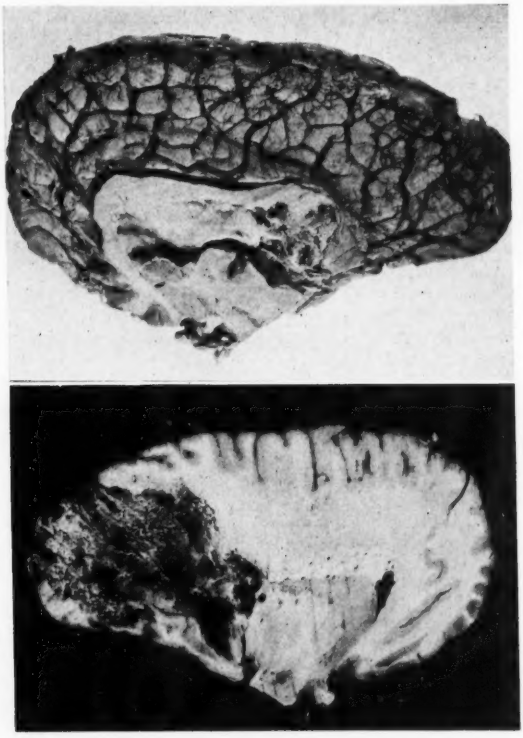

FIG. 3. A large right occipital glioblastoma with extensive invasion of the splenium of the corpus callosum as seen on sagittal section. The tumor spread even further to involve the left occipital lobe.

The temporal lobes have only feeble interhemispheric connections through the anterior commissure and tapetum, and but a small isthmus connecting them with the parieto-occipital region. There is, in our series, no instance of a glioblastoma of the temporal lobe that has crossed over to the opposite hemisphere. Except for local invasion of vital basilar regions, the right temporal lobe would theoretically offer the most suitable conditions for extensive resection.

Of the 7 cases in which callosal invasion could not be proved grossly,

in only one, a left frontal tumor, a possibility of extensive resection might have been successful since the deeper structures were not grossly involved. However, in the other 6 cases (2 left temporal, 2 right temporal, 1 left parietal, and 1 left parieto-occipital) had a complete excision of the tumor been accomplished, a severe crippling of the patient would have resulted, as there was direct invasion of the speech centers, basal ganglia or internal capsule in each case.

SUMMARY AND CONCLUSIONS

This evidence of the high frequency of gross interhemispheric extension of glioblastoma multiforme through the corpus callosum (75 per cent in our autopsy series of 28 consecutive cases) supports the rationale of conservative treatment (decompression, deep x-ray therapy) of this malignant glioma, except in the case of superficial (lateral to the temporal ventricular horn) right temporal sites.

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EARLY COMPLICATIONS FOLLOWING PENETRATING WOUNDS OF THE SKULL

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AN ANALYSIS of 14,000 battle wounds treated in 5th Army hospitals in the year 1944 disclosed that 6.17 per cent involved the head (exclusive of maxillo-facial injuries). One third of these were classified as intracranial† while the remainder were scalp wounds. At the same time 24.4 per cent of all deaths in these institutions were attributed to brain injuries. The vast majority of these patients were hopelessly injured and died within a short time after reaching a forward hospital. Of those patients with penetrating skull wounds who survived to reach a general hospital in the Zone of Communications, another 3.7 per cent succumbed. For the most part these deaths were ascribable to complications of wound healing. These disorders occurred not infrequently and even though not fatal in the majority of cases, entailed further damage to neural tissue and prolongation of convalescence. It is to describe our experiences in dealing with the problems that arose during the early postoperative course of 426 cases of penetrating skull wounds that this paper is written.

Since morbidity and mortality in military surgery vary according to the echelon, and to the tactical situation, and since weather, topography and evacuation are factors that must always be considered, the following facts are pertinent. The patients whose cases form the basis of this report were wounded in the Tunisian, Sicilian and Italian campaigns. The majority were operated upon in evacuation hospitals, situated 5 to 20 miles behind the front lines. After 3 to 10 days they reached general hospitals, consecutively located in Oran, Bizerte, Rome and Leghorn, where they came under the care of the authors. Evacuation was by ambulance, air, rail or hospital ship. In general the trip appeared to have upset them very little, save those individuals with elevated intracranial pressures. During the period in which this study was made, the skill and experience of forward neurosurgeons greatly increased, with the result that debridements were carried out more thoroughly. Penicillin was used routinely (25,000 units intramuscularly every 3 hours) beginning in the Spring of 1944.

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† Major W. R. Pitts reported 108 (1.03 per cent) penetrating brain wounds which came to operation among 10,455 battle casualties admitted to the 38th Evacuation Hospital. Shearburn operated upon 139 (1.6 per cent) patients with similar lesions among 8,660 battle casualties at the 8th Evacuation Hospital; an additional 28 patients were desperately ill on admission and died without operation.

SUPERFICIAL INFECTION

Superficial infection occurred in approximately 19 per cent of the cases in 1943; the incidence was approximately 16 per cent or significantly unchanged in 1945. In this category we have included infections that extended no deeper than the dura, wound dehiscence, and necrosis of even mild degree (in other words any condition less than *per primum* healing). For the most part they were mild, localized processes, usually associated with too much tension or too heavy silk. Removal of sutures and sometimes the application of hot wet dressings sufficed to clear them up within a few days. Rapid healing was not always the case, however, and if the dura had been left open grave complications sometimes ensued. If the wound were large, and particularly if the debridement had not been thorough, it often filled with dirty, purulent granulation tissue and required weeks to heal.

The *types of incision* employed and the tensions under which they were closed were factors of considerable importance. Even mild wound necrosis or local dehiscence usually led to superficial infection. Where practical the simple vertical or curvilinear incision was most satisfactory from the standpoint of healing. Quite often so much scalp was destroyed that direct closure, particularly after debridement, could not be carried out. To effect approximation under these circumstances was not easy. Methods commonly employed were the "tripod" incision or its Isle of Man modification, one or more relaxation incisions or a large flap. The latter sometimes included the scalp defect in one limb or, if small, in its center.

Tripod incisions, while affording a convenient and rapid exposure, have, in our experience, not healed well. In one series of 26 cases, necrosis of the apex of one or more flaps, wound separation or infection occurred in 18. Sometimes a multangular wound made such an incision unavoidable. When employed, great care must be exercised in handling the flaps to avoid bruising their apices. The flaps should be sufficiently long for the limited elasticity of the scalp to permit stretching over the defect without undue tension. The narrowest angled flap should be on the side of the best blood supply, while constricting sutures at the apices should be avoided.

If relaxation incisions or pedicle flaps are used, it is well to leave the pericranium intact and to cover the defect with a split thickness graft immediately. Grafting requires but a few additional minutes' work and goes far to insure primary healing. Failure to do so led to local infection in several instances, thus endangering the primary wound.

S-shaped incisions, in which the wound was included in the central limb, proved very satisfactory. Here again it is well to point out that they should be generously long, for the scalp has but limited elasticity, and it is desirable to distribute the tension over a wide area.

DEEP INFECTION

Deep infection occurred in 72 or 16 per cent of our 426 cases. Local purulent cerebritis progressed to abscess formation in 58 cases, and resulted

in cerebral fungi in 32, and in meningitis in 25. Of these patients, 15 died. One patient succumbed from a reaction to mismatched blood. There were no deaths from other causes in this group. The mortality rate was therefore 22 per cent for those patients who developed deep infection or 3.7 per cent for the group as a whole. In the light of our experience it would appear that several of these deaths might have been prevented had infection been recognized earlier.

A summary of our observations follows: Infections occur within the cerebral wound approximately 10 times more frequently about roentgenologically retained bone fragments than in their absence. Their presence is a measure of the thoroughness of wound debridement. Together with clots and pulped brain tissue they offer a splendid medium for the growth of the bacteria inevitably carried into the wound. If the process is not too extensive and bacterial contaminants not too numerous or too virulent, healing can and often does take place spontaneously. However, in a series of 49 cases in which bone fragments were retained, 20 developed deep infection, whereas in a similar group of 45 cases in which the removal had been complete (as determined by roentgenograms) but two infections occurred.[1]

The pathologic process is not strictly comparable to that encountered in a brain abscess of otitic origin. Here, in addition to the neural tissue necrosed by trauma and bacterial toxins, are the added factors of extravasated blood, of indriven bone and foreign bodies. We believe that these factors must be taken into consideration in treatment.

The *bacterial flora* of both deep and superficial infections was made up largely of skin inhabitants of relatively low virulence. In another paper[1] one of us reported the results of bacteriological studies in 25 infected head wounds. Of 39 organisms recovered, 23 were hemolytic or non-hemolytic staphylococci. It is noteworthy, however, that of 9 strains of bacteria isolated from the wounds of 6 patients with meningitis, 6 were streptococci (3 aerobic non-hemolytic, 2 aerobic hemolytic and 1 anaerobic hemolytic) while but 3 were staphylococci (all aerobic non-hemolytic).

Diagnosis of Deep Infection. The early manifestations of deep infection are frequently mild and easily overlooked. A well-healed pulsating scalp flap combined with normal temperature, normal white blood count, and the absence of cervical rigidity may give one a false sense of security. Headaches, at first slight and later increasing in frequency and severity, have been the most constant manifestations of smouldering infection. Head pains that come on suddenly or awake the patient from sleep are particularly significant. Later on the flap may become tense, the discs choked, neurological changes progress and signs of increased intracranial pressure appear. The roentgenological demonstration of retained bone fragments, particularly if in a cluster, then becomes highly significant, for it was about these that 90 per cent of the abscesses developed. On 5 occasions a retained metallic fragment was observed in successive roentgenograms to have changed position, and in all but one instance was found to lie free within an abscess

cavity. In the fifth case it was found in a cerebellar hematoma. There was no instance of loose metallic fragment in a ventricle in this series of patients.

The manifestations of meningeal irritation or infection are well known. When arising as the result of spread from the deep infection it was often attributable to direct extension into a ventricle. The subarachnoid spaces were, as a rule, well sealed for approximately 1 cm. about the edge of the cortical wound.

The presence of more than a few cubic centimeters of necrotic brain tissue, even though not infected, may give rise to irritation and local edema with increased intracranial pressure, much in the manner it sometimes does following any extensive resection of cerebral tissue. It is usually impossible preoperatively to differentiate it from an early infection. Prompt alleviation of symptoms may be expected to follow its removal.

Treatment. Delayed debridement or redebridement of infected brain wounds was successfully employed towards the end of the Tunisian campaign by Schwartz.[2] We have repeatedly confirmed the efficacy of the procedure. Redebridement of wounds elsewhere in the body, including compound fractures, has likewise proved a very useful operation where early surgery had been inadequate or totally lacking. The presence of dead tissue is of considerably more danger than the breaking of newly formed "barriers of resistance," save when the last boundary before a ventricle or mesothelial-lined space is concerned.

While it might appear desirable to postpone operative intervention until abscess formation has occurred, it should be pointed out that encapsulation, if occurring at all, is sometimes quite tardy and that much mischief can be done by the infection in the interim. Further destruction of brain tissue, the development of cerebral fungus or fatal meningitis may take place before a frank abscess is formed. The following case from the Bizerte series is illustrative and represents our lack of appreciation of these facts at that time:

Case 1. A young British infantryman was struck in the right frontal region by a shell fragment. There was slight weakness of the left face and arm. The wound was debrided and closed without drainage on the same day at an evacuation hospital; 48 hours later some bloody fluid was said to have escaped through the wound.

He was admitted to the Head Center 4 days after injury, at which time he was alert, comfortable and cooperative. The wound was well healed, the flap soft and pulsating. There was no fever or tachycardia. Roentgenograms disclosed several small bone fragments beneath the skull defect in the right frontal region and a large metallic foreign body just to the left of the midline in the frontal region. The following day his neck became slightly stiff and the flap bulged. Several cc. of serosanguineous fluid containing flecks of necrotic brain tissue were aspirated from beneath the flap. Smears of this material revealed no bacteria but cultures could not be made since our laboratory had not been set up completely at the time. The next day the bone chips were removed but the bullet, which lay beyond the falx, was not touched. Death from meningitis took place 2 days later, or 9 days after wounding.

Autopsy disclosed pus about the metallic fragment but no sign of capsule formation (Fig. 1). Direct extension into the ventricle was not demonstrated in this case.

Comment. Had this wound been thoroughly debrided and the huge

metallic foreign body removed at the first operation, or by us at our first opportunity, it is altogether likely that this catastrophe would have been avoided. The history of the next patient exemplifies the favorable course that may be anticipated after adequate redebridement.

Case 2. An American infantryman incurred a penetrating wound of the right frontal lobe when struck in the right forehead by a shell fragment at 2200 hrs. on 26 October 1944. The

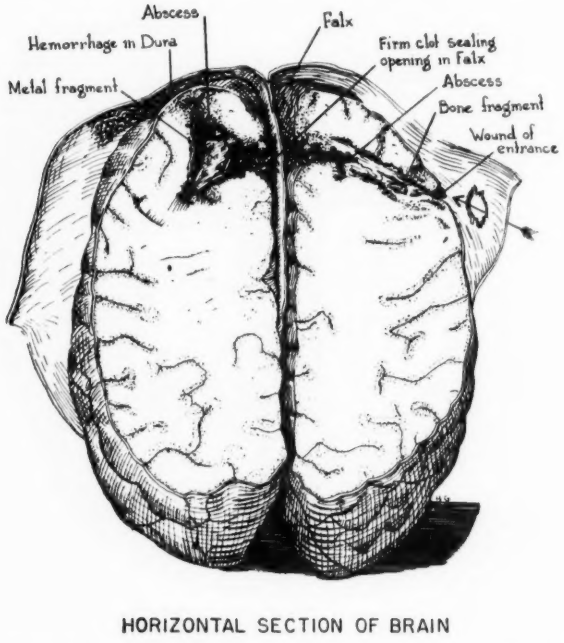

FIG. 1. Case 1. Drawing from autopsy specimen showing inadequately debrided penetrating wound of brain. Death from meningitis occurred on the 9th day following injury.

wound was debrided 30 hrs. later, at which time bone fragments, a few clots and some necrotic brain tissue were removed. The dura was not closed. His immediate convalescence was apparently quite satisfactory, neurological changes having remained minimal.

When seen in the Head Center 10 days later he was alert, cooperative and intelligent. He complained of mild pains in both eyes and of precipitate urinary and fecal incontinence. The Z-shaped incision was well healed but, though pulsating, seemed a little full. Early bilateral papilledema was present and the neck was slightly stiff. Roentgenological examination of the skull disclosed a 5×4 cm. defect in the right frontal bone, with radiating linear fractures, one of which entered the cloudy right frontal sinus. A bone fragment, $13 \times 6 \times 5$ mm., was observed 5 cm. beneath the defect and 2 cm. to the right of the midline. A $17 \times 6 \times 4$ mm. metallic foreign body was present in the midline, approximately in the region of the midportion of the corpus callosum.

The wound was reexplored on 9 November 1944 under general anesthesia. Immediately beneath the flap several cc. of necrotic brain tissue of mushlike consistency were found and

removed. Cultures taken from this material yielded non-hemolytic Staphylococcus albus and E. Coli. Following removal of the large bone fragment in the depth of the wound there was an escape of several cc. of watery fluid containing particles of pulped brain tissue. The shaggy walls of the wound were redebrided back to viable looking tissue. A hematoma-filled tract extended along the right side of the falx and apparently led back to the metallic fragment. The clot was removed no further back than the genu of the corpus callosum, since the tract from there on was narrow and exposure difficult. The shell fragment was not removed. Penicillin (10,000 units in 5 cc. of saline) was left in the cavity. The galea and skin were closed in layers with fine silk without drainage.

Convalescence was prompt and uneventful. The incision healed *per primum*. Urinary and fecal incontinence had disappeared by the 9th day.

Drainage of deeply infected brain wounds, as well as of frank abscesses, has been very unsatisfactory in our hands. If redebridement had been done satisfactorily a drain was not necessary, and if not, it did not suffice. On the other hand, extensive purulent granulation tissue beneath the scalp calls for a soft drain of some kind for a few days.

MENINGITIS

Meningitis occurred in 25 or 6 per cent of our cases; there were 9 deaths. Organisms cultured from the wounds (but not always from the cerebrospinal fluid) were: aerobic hemolytic streptococcus, 3; anaerobic hemolytic streptococcus, 1; pneumococcus, 2 (one type 29, the other unknown); aerobic non-hemolytic streptococcus, 3; aerobic non-hemolytic Staphylococcus albus, 5; A. aerogenes, 2; B. Coli, B. subtilis and diphtheroids, each 1. We believe that in those cases in which the infection has passed from the infected wound into the ventricle or subarachnoid space, one should, providing the patient's condition permits, redebride the wound as early as possible, and at the same time vigorously employ chemotherapy. This applies particularly to incipient meningitis. If, on the other hand, infection has entered through a fracture of a paranasal sinus, it is better to rely solely on chemotherapy in the beginning, since the results of this method can be expected to be good, and second, the operative closure of the dural laceration would be difficult and hazardous in a very sick man whose brain was edematous. This latter procedure may then be done at a future date when the patient is in better condition.

As an example of the first problem (meningitis from an infected wound) the following case may be cited:

Case 3. An American soldier received a penetrating wound of the right temporal lobe from a shell fragment on 4 February 1944. Six hours later debridement and closure with drainage was performed. His initial convalescence was said to have been satisfactory. The drain was removed after 2 days. However, the wound broke down.

When he entered the Head Center 2 weeks later headaches had become a prominent symptom. The flap was bulging and tense; pus was draining from the posterior end of the incision and spinal fluid from the anterior. Four days later his neck became quite stiff. Lumbar puncture revealed the presence of 560 white cells in the spinal fluid, of which 94 per cent were polymorphonuclear leucocytes, while cultures disclosed non-hemolytic Staphylococcus albus.

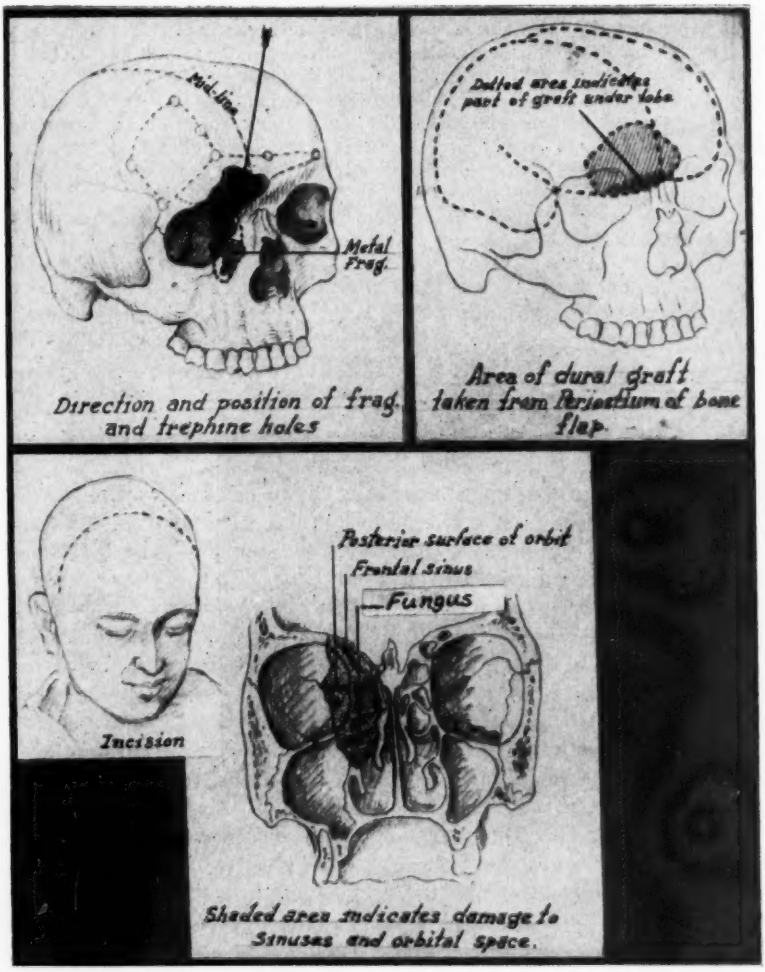
Metal frag.
Direction and position of frag. and trephine holes
Dotted area indicates part of graft under lobe
Area of dural graft taken from Periosteum of bone flap
Posterior surface of orbit
Frontal sinus
Fungus
Incision
Shaded area indicates damage to Sinuses and orbital space.

FIG. 2. Case 4. Diagrammatic drawings showing position of wound in orbital ridge and in frontal and ethmoidal sinuses, the latter having been filled with a small fungus.

Redebridement of the wound and removal of several retained bone fragments, together with intensive sulphathiazole therapy, were followed by healing and prompt subsidence of the meningitis. Cultural studies of the wound revealed the presence of non-hemolytic Staphylococcus albus and A. Aerogenes.

Rhinorrhea, if not profuse, may pass unnoticed or the patient, in view of his other woes, may not consider it worthy of complaint. The soldier whose history follows was wounded in the battle for the Gothic Line, entered

the Head Center during a period of great rush, and the existence of the fistula was not suspected until meningitis had set in.

Case 4. A 28-year-old infantryman received a penetrating wound of the right frontal lobe when struck by a shell fragment at 0415 hrs. on 7 October 1944. The fracture extended into the right frontal and ethmoidal sinuses. The wound was debrided and closed at an evacuation hospital 29 hrs. later. Convalescence was prompt, and the wound healed *per primum.*

When he reached the Head Center almost a month later he was ambulatory and without

FIG. 3. Case 4. Photograph showing healed incisions approximately 2 weeks after operation. The right eye had been enucleated at the initial debridement.

complaints. No neurological changes were observed. On 16 November 1944 meningitis suddenly set in. Then, belatedly, we obtained the story of rhinorrhea. It was said to have been present almost constantly until the previous day, when it had ceased. Pneumococcus, type 29, was recovered from the spinal fluid. He was alarmingly ill, but responded promptly to sulphathiazole and routine intramuscular penicillin (25,000 units every 3 hours).

On 7 December 1944 a right frontal craniotomy was performed under ether by Captain W. B. Weary. A large encapsulated subdural hematoma overlaid the frontal lobe; the lower portion of the latter had herniated through the defect in the cribriform plate into the right nostril (Fig. 2). A small aerocele was found in this fungus when excised. After removing the hematoma the dural defect over the frontal and ethmoidal sinuses was repaired with fascia lata. After operation an extradural hematoma developed, the removal of which was followed by rapid recovery (Fig. 3).

OSTEOMYELITIS

Osteomyelitis of the spreading type, which sometimes follows frontal sinus infection, was not observed in this series of cases, and but one in-

stance of a relatively mild nature has come to our attention elsewhere. A low-grade inflammatory reaction in the edges of an infected craniotomy was commonly seen, but seldom gave trouble after its removal by rongeur during the redebridement of the wound.

CHEMOTHERAPY

In order to assess the value of sulphonamide or penicillin therapy in penetrating skull wounds, comparable series of cases must be studied. We have observed no such group in which neither drug was employed. The incidence of deep infections during the period in which sulphonamide therapy alone was practised was 37, or 23 per cent of 158 cases, while following the introduction of penicillin therapy in the Spring campaigns of 1944 the rate fell to 35, or 13 per cent of 268 cases. While this is apparently* an improvement, it is felt that at least two other factors must share the credit with penicillin, namely, better debridement and the more generous use of whole blood replacement therapy. The value of penicillin and/or sulphonamides in arresting the invasion of certain bacteria is widely recognized; it is equally important to bear in mind the inability of either agent to sterilize a wound containing necrotic tissue, blood clot or foreign matter. Furthermore, in all fairness to Mother Nature, it is to be recalled that well-debrided wounds in the brain or elsewhere usually heal well without the benefit of any chemotherapy.

Sulphonamides have not been used locally in brain wounds because of their irritating effects. During the past year it has been customary to instill a solution of penicillin (10,000 units in 10 to 15 cc. of normal saline) in the wound after completion of debridement. It is not possible to prove the value of this particular procedure, since these patients received penicillin intramuscularly as well. We have observed no ill effects from its local use, although on more than one occasion mild irritation of the meninges appeared to have resulted from repeated intrathecal injections.

CEREBRAL FUNGUS

Cerebral fungus occurred in 32 cases, an incidence of 7.4 per cent. In another paper ("Cerebral fungi complicating penetrating brain wounds": to be published) we pointed out that this condition is to be looked upon as a complication of wound healing, and that treatment must be directed primarily at the cause rather than at the fungus itself. In this series fungi that appeared following wound debridement readily fell into two groups.

The first, which we termed *benign*, was made up of 13 instances of local cerebritis associated with superficial wound infection and an open dura. The herniae grew slowly, were seldom large, shaggy or friable, nor were they associated with high intracranial pressure nor progressive neurological

* Since the observed difference 10 is less than twice the P.E. (5.81), the probability of this occurring by chance is about 1 in 4 and the significance is therefore highly questionable.

changes. None was accompanied by deeper infection. There were no deaths in this series. Hot wet dressings were employed but otherwise no special treatment was required, since the natural tendency was toward healing and retraction of the hernia. In but one early case was the mass excised. It was 3 to 4 cm. in diameter, was found to be undergoing fibrosis and would in all probability have subsided spontaneously. Retained bone fragments were observed in but 4 individuals and were removed from 3. When located superficially it is probably better to remove them, since they so frequently harbor infection. The following case, reported in brief, illustrates the problem of benign fungus.

Case 5. A 23-year-old infantryman suffered a severe penetrating wound of the right parietal lobe with complete left hemiplegia on 25 October 1944. Indriven bone and metallic fragments, blood clots and pulped brain tissue were removed at the time of wound debridement 29 hours later. The dura was not closed.

When admitted to the Head Center 11 days afterwards the hemiplegia remained complete. The wound was gaping widely and bathed in pus; in its center was a small herniation of cerebral tissue, approximately 1 inch in diameter and ½ inch in height. It was pulsating fairly well. He was afebrile, alert, cooperative and complained of only very mild headaches. There was no papilledema. Roentgenograms disclosed no retained bone or metallic fragments. There being no evidence of markedly elevated pressure, meningeal irritation or retained bone fragments, it was assumed that the fungus was brought about by the local cerebritis associated with a superficial wound infection in the presence of an open dura.

Hot wet dressings were applied to the wound; sulphathiazole and penicillin therapy were instituted. The local infection subsided, the fungus gradually receded and within 3 weeks the wound had healed. During this time the hemiplegia began to clear up.

Nineteen patients in this group developed cerebral herniations associated with increased intracranial pressure. In each instance deep infection, hematoma or rarely a large mass of necrotic brain tissue was responsible. Once started on their way through open dura these fungi grew rapidly, were shaggy and bled easily. Evidence of increased intracranial pressure and progressive destruction of neural tissue was readily found. We termed these *malignant* fungi. A hernia of this type may follow the intense cerebritis of a virulent local wound infection, but we did not encounter it in this series of recently debrided war wounds. In every instance early surgical intervention was required. Accurate preoperative localization of retained bone and metallic fragments by stereoscopic roentgenograms was essential. Removal of the necrotic portion of the fungus often served to reduce pressure locally and occasionally uncovered a sinus leading down to an abscess. Six patients died while under our care and three others may have succumbed subsequently, since the military situation required their evacuation while they were still quite sick. In each fatal case the underlying abscess had been overlooked or its evacuation too long delayed.

As an example of malignant fungus improperly treated the following case is recorded:

Case 6. An infantryman incurred penetrating wounds of the left parietal lobe, right ear, right mandible and right groin from a machine pistol on 13 October 1943. Records of the initial operations were lost but it was known that meningitis had set in while on a hospital

ship between Italy and Tunisia. At that time a lumbar puncture had revealed gram-positive diplococci in the spinal fluid. Improvement followed intensive sulphadiazine therapy.

On arrival at the Neurosurgical Center 11 days after injury he was semi-stuporous and inarticulate. There were complete right hemiplegia and early bilateral papilledema. A tense, shaggy cerebral fungus, 2 inches in diameter and 1 inch in height, was present in the left parietal region. Roentgenograms disclosed many retained bone fragments in the left parietal lobe and a few more, together with a piece of metal, in the left temporal lobe. Sulphonamide therapy was continued. Within the next 3 days he improved somewhat and spoke a few words but remained almost completely apraxic.

On 28 October 1943 the fungus was excised and the wound in part redebrided. Several small bone and metallic fragments were removed. An abscess beneath the parietal defect, which contained perhaps 5 cc. of pus, was evacuated. Improvement was but slight and within 4 days the spinal fluid pressure had risen again to approximately 400 mm. of water and meningitis developed. Spinal fluid cultures revealed aerobic non-hemolytic streptococci and Staphylococcus albus. The fungus reappeared, thrusting outward the original abscess capsule. It was reexcised and the parietal and temporal lobes were unsuccessfully explored for abscess with a ventricular needle. The following morning another exploratory tap disclosed a left temporal lobe abscess, from which approximately 20 cc. of pus were evacuated. It was too late, however, and he died the same day.

Autopsy revealed a poorly encapsulated abscess located principally in the temporal lobe but extending into the frontal white matter as well. It had formed in the terminal portion of the principal missile tract. Rupture had occurred into the lateral ventricle, the ependyma of which was covered with shaggy exudate. One or two small bone fragments were incorporated within the abscess. The metallic foreign body in the temporal lobe was surrounded by a tiny independent abscess about ½ in. beyond the larger cavity.

Comment. Within a few days after the first redebridement, mounting intracranial pressure indicated that the seat of the trouble had not been found. At that time abscess was the diagnosis, and one should not have given up after unsuccessful tapping. Abscesses may be difficult to find. It should be recalled that the missile and the bone fragments seldom follow the same tract, and that abscess occurs much more often about bone fragments. A ventriculogram should have been made in this instance.

In the following case the abscess was readily evacuated with prompt relief.

Case 7. A 31-year-old infantryman was struck in the left frontal region by a rifle bullet on 16 October 1943 in Italy. The wound was debrided, the missile removed, and closure effected the following day at a field hospital. A few days later at another institution the wound was observed to have broken down and a fungus was evident. The amount of sulphonamide administered was unknown, and penicillin was not available.

On arrival at the Head Center on 25 October 1943, 8 days after the initial operation, the wound was found to be draining pus and a tense, shaggy, non-pulsating fungus was present. Neurological examination revealed mild right hemiparesis and early papilledema. Mental processes were slow, but he was rational, cooperative, and complaining of little headache. There was little fever, but the neck was slightly stiff. Roentgenograms revealed several retained bone fragments a short distance beneath the defect in the posterior portion of the left frontal bone.

Under novocaine anesthesia the fungus was excised flush with the dura. A mass of necrotic tissue and bone fragments was found beneath associated with a partially encapsulated abscess. Following evacuation of the latter and secondary debridement of the wound, the scalp was closed loosely about a drain. Wound cultures revealed aerobic hemolytic Staphylococcus albus, and aerobic hemolytic streptococcus. His highest recorded cerebrospinal fluid pressure

was 200 mm. of water, and a cell count of 330, of which 40 per cent were polymorphonuclear leucocytes. Convalescence was uneventful and his neurological recovery complete.

HEMATOMA

Hematoma within the cerebral wound was the rule, whereas large subdural and particularly large extradural extravasations were unusual. Among a group of 333 cases, subdural hematomas were found 7 times; extradural and subtentorial clots were even less common. In this echelon one encountered the overlooked or recurrent clot, which may have occurred at any point along the missile tract. Major J. Shorstein of the R.A.M.C. has drawn attention to the huge intracerebral clot that sometimes follows the small missile wound. The following case illustrates this problem, which is more commonly encountered by the forward than the base surgeon.

Case 8. A German prisoner was brought to the hospital 2 days after having been wounded in the battle for Cassino. He was very drowsy and could give little account of what had happened to him. Partial motor aphasia and right facial weakness were obvious. Close examination of his head revealed a tiny pin-point wound in the left temple. Roentgenography disclosed a metallic fragment, approximately 2 mm. in diameter, within the temporal lobe. Exploration of the wound disclosed a subcortical hematoma of 3 to 4 cm. diameter, the removal of which was followed by prompt recovery.

The more commonly encountered complication is that of overlooked or recurrent hematoma within the wound. If the ventricle has been traversed, either blood or infection may set up an alarming reaction. The next case offers an unusually interesting example of such a problem.

Case 9. A 24-year-old infantryman was rendered unconscious by a mortar shell fragment which entered the right frontal lobe and came to rest in the left temporal region. When subsequently examined at an evacuation hospital he was drowsy and confused but quiet. The temperature was 100.6°F., pulse 60, respirations 18 and blood pressure 130/60. Speech was slow and hesitant but he could name test objects. The right frontal wound was 1.5 cm. in diameter; 33 hrs. after injury it was debrided down to a perforation in the falx, the tract having been found not to traverse the right ventricle. Numerous bone chips, clot and a considerable amount of pulped brain tissue were removed, the dura was repaired with a pericranial graft, and the scalp closed in layers without drainage. Penicillin (10,000 units) was instilled into the wound and subsequently administered by intramuscular injection as well.

Postoperative improvement was but slight and temporary. Within 2 days fever, cervical rigidity and increasing stupor were evident and he was evacuated to the Head Center.

He arrived conscious but slightly drowsy, with a rigid neck. The temperature was 104°F., pulse 98 and respirations 28. There was mild left hemiparesis. The lumbar spinal fluid was grossly bloody and under a pressure of 500 mm. of water. (No organisms were subsequently grown from this material on culture.) The craniotomy wound was well healed. Roentgenography disclosed a large metallic fragment in the left temporal lobe 3 cm. above the petrous pyramid. Two bone chips were visible just to the left of the midline between the metallic foreign body and the right frontal bone defect. By the following day he was quite stuporous, the temperature had reached 105°F., and it was observed that he perspired only on the left face and trunk.

Operation: 20 July 1944 (Capt. W. B. Weary). Under ether anesthesia, a 6 cm. opening was made in the squamosa of the left temporal bone. At one point the dura was obviously bruised on its inner surface. Several small clots and bits of pulped brain tissue marked the posterior tract of the missile from the point of ricochet. After cleaning out this path and removing the fragment, the tract was followed forward. Numerous clots of rubbery con-

sistency were evacuated and the anterior horn of the left ventricle encountered. Both in this region and in the ventricle itself was an extensive hematoma. This was removed by washing and suction, as were the bone fragments which lay nearer the falx. The foramen of Monro was clearly in view. Despite good hemostasis and repeated irrigation of the tract the brain remained under tension. Consequently the wound was closed, and the right frontal craniotomy reopened. Two hematomas were found and removed, the second being located just to the right of the falx. The brain was then quite slack. The dura and scalp were resutured in layers without drainage.

His postoperative course was rather stormy for the first 48 hrs. and the temperature was not normal for 9 days. There was mild bilateral facial weakness, but although speech was slow and hesitant, it was fairly complete. He was able to be out of bed 2 weeks after the operation. Reports several months later from the Zone of Interior indicated that he was greatly improved and was talking quite satisfactorily.

While subdural hematoma may be associated with a penetrating wound, it can also follow a glancing blow. As in blunt injuries, it may be situated beneath the point of impact or at some distance. The following case was of interest in that the extravasation of blood was in the posterior fossa.

Case 10. A 20-year-old infantryman was struck in the right ear by a sniper's bullet on 17 July 1944. The huge gutter wound, which extended through the external ear, over the mastoid process and through the muscle in the right suboccipital region, was debrided and partially closed.

When admitted to the Head Center 3 days later he complained of throbbing, right-sided headaches, counter-clockwise vertigo and diplopia. Neurological examination revealed bilateral papilledema, deafness in the right ear, weakness of both external recti and bilateral ataxia and atonia of the arms and legs, the left having been more affected than the right.

At operation (Capt. W. B. Weary) a large subdural hematoma was found covering both cerebellar hemispheres. Following its removal he recovered rapidly, although slight ataxia was still evident when he was evacuated 3 weeks later. In a letter written from the United States several months later he indicated that he was symptom free.

EPILEPSY

Convulsions occurred not infrequently between the time of wounding and debridement. They were quite unusual in the early postoperative period (1 to 2 months in our echelon). When they appeared within the first 2 to 3 weeks after operation they quite often signaled the presence of an abscess, a hematoma or a pocket of necrotic tissue in the cerebrum. In the series of 100 penetrating skull wounds referred to[1] but 5 patients had fits postoperatively (exclusive of cases of meningitis). "All of these were associated with some degree of motor weakness, and were Jacksonian in type." Three of these patients were subsequently found to have abscesses. Convulsions of alarming severity were initiated by a small infected hematoma near the motor cortex of the patient whose story follows:

Case 11. This soldier received a penetrating shell fragment wound in the left temporoparietal region on 20 July 1944. Six hours later the wound was debrided at an evacuation hospital. The cerebral wound was approximately 2×2×3 cm. in size. Satisfactory hemostasis was said to have been obtained.

On reaching the Head Center 4 days later he was seemingly in excellent condition, save for mild left-sided headaches. The wound was well healed and no neurological changes were observed. Two days afterwards he suddenly vomited and became stuporous. An hour later

a second vomiting attack was followed by violent right-sided convulsions. These began in the right face, spread to the right hand, arm and leg; they persisted, and 1½ hrs. later became generalized. Continued cyanosis and a thready pulse were cause for concern.

The wound was reexplored under ether anesthesia and found to contain a mass of hematoma and necrotic brain tissue. Cultures from this material yielded hemolytic Staphylococcus albus. Thorough wound toilet and tight closure were followed by rapid recovery. At the time of evacuation a month later there were no neurological abnormalities and he had had no more fits.

DURAL CLOSURE

Dural closure has long been a moot point in the treatment of penetrating wounds. This is but natural when the majority of wounds heal satisfactorily whether or not the dura is sutured. It has been argued that it should be left open to permit spontaneous drainage if infection occurs. In our experience deeply infected brain wounds have not drained satisfactorily and redebridement has usually been necessary. On the other hand, we have on at least four occasions observed the deeper wound effectively shielded from superficial infection by a dura closed either by suture or graft. In an equal number of wounds of the spinal dura, closure by suture or graft prevented inward extension of epidural infection. Another possible advantage may accrue in deeply infected brain wounds, whereby the formation of a fungus is prevented or delayed. We believe that where practicable the dura should be closed; if simple suture is not possible then a graft of living tissue (pericranium, temporal fascia or fascia lata) is advisable. That a living graft imposes little additional burden on the wound is indicated by the following comparison. In one series of 50 cases of penetrating brain wounds in which no graft was employed, there were 7 deep and 6 superficial infections. Among an equal number of similarly wounded individuals whose dura had been patched there were 9 deep and 6 superficial infections.

COMMENT

The vast majority of individuals whose penetrating skull wounds entailed irreparable damage to vital parts of the brain died on the battlefield. In a series of 1000 autopsy examinations on soldiers who succumbed before reaching an aid station, Captain W. W. Tribby of the 2nd Medical Laboratory, 5th Army, found that 133 had only head wounds, while a total of 401 had incurred head wounds of sufficient severity to have caused death. For the year 1944, 24.4 per cent of all deaths in the 5th Army field and evacuation hospitals were attributed to head injuries. The great majority of those patients who lived to reach general hospitals 4 to 10 days later had brain damage that was compatible with survival. The chief hazards to be faced are the complications of wound healing, which are for the most part overlooked, or recurrent hematoma, masses of necrotic brain tissue and infection; most of these stemmed from incomplete debridement.

Those of us who have had the privilege of operating in forward medical installations during heavy fighting realize all too well the difficulties under which surgeons must work. These are well known and require no elaboration,

save perhaps on two points. The first is the need for better roentgenograms. The incidence of deep infection has been shown to vary directly with the thoroughness of wound debridement. The number of demonstrably retained bone fragments may be taken as one measure of this. We feel strongly that better work could be done if stereoscopic roentgenograms were available to the neurosurgeon, for flat plates, even when they happen to be clear, leave much to be desired. The equipment is available and the x-ray department is usually an hour ahead of the operating room during rush periods; but seldom have these views been made.

A second factor of no little importance is that of fatigue. After a few days of working under heavy pressure the neurosurgical team becomes very, very tired. When the tactical situation has permitted combining the teams of two evacuation hospitals in the more forward institution and triaging all head cases there, it appeared that more and better work was done. When operating on a 12-hour shift the teams could carry on day after day without undue fatigue. It is realized that this arrangement will not always be possible but when it is, we believe that the combination of two teams is of distinct advantage.

SUMMARY AND CONCLUSIONS

The early complications that followed 426 penetrating skull wounds of the Tunisian, Sicilian and Italian campaigns have been studied. Superficial infection, local skin necrosis or dehiscence took place in 74 cases. Tripod incisions were associated with faulty healing in the majority of instances in which they were employed. When possible, their use should be avoided. By rotating flaps or making relaxation incisions practically all scalp defects can be covered by full thickness skin. The bed from which the flap has been moved should be covered by epithelium at once. If suture is not feasible because of tension, a split thickness graft serves well.

Deep infections developed in 72 cases (16.8 per cent), in the great majority of which debridement was demonstrably incomplete. In this group there were 58 abscesses, 32 cerebral fungi and 25 cases of meningitis. There were 16 deaths (3.7 per cent for this echelon), only one of which was not due to infection. The diagnosis and treatment of these complications were discussed and illustrative cases cited. The value of redebridement and late debridement was stressed.

The ability of sulphonamides and/or penicillin to check the invasion of certain bacteria is unquestioned. On the other hand, the power of either to prevent or to arrest bacterial growth in a wound, particularly if the latter be incompletely debrided, is doubtful. Although the incidence of deep infection diminished from 23 to 13 per cent after the use of penicillin became routine (Spring of 1944), it is to be recalled that with greater experience and skill debridements were done with more thoroughness during this latter period. At the same time the more generous use of whole blood made it possible to maintain patients in better general condition. The value of thor-

ough debridement in the prevention of deep infection was shown in an earlier study by one of us (E.H.C.). In a series of 94 cases it was found by stereoscopic roentgenograms that 45 had had all indriven bone fragments removed, while in 49, some part or all of these had been left behind. Twenty-two deep infections occurred in the latter group and but 2 in the former. Penicillin was not in use at that time. From the above observations it appears that most brain wounds will heal without deep infection if adequately debrided, but if this is not done a number of infections will occur whether or not penicillin or sulphonamides are employed.

Cerebrospinal fluid fistulae are conducive to meningitis. Penetrating craniocerebral wounds that involve the paranasal sinuses usually require dural repair. Should meningitis supervene beforehand, it is advisable to treat it vigorously with both sulphonamides and penicillin. As soon as the infection has subsided and the intracranial pressure has returned to normal, the dural rent should be closed.

Hematomas occurred most frequently along the missile tract, particularly in its distal portion. Necrotic cerebral tissue, with or without infection, often accompanied the clot and was a common cause of elevated intracranial pressure. Subdural and large extradural hematomas were unusual.

Epilepsy was quite uncommon during this early period and when it appeared often signaled the presence of an abscess, a hematoma or a focus of necrotic tissue.

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THE USE OF CURARE IN THE TREATMENT OF SPASTIC PARALYSIS

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THE PURPOSE of this report is to direct attention to the usefulness of curare in the management of patients suffering with spastic paralysis, and to describe and discuss an heretofore unreported technique of administration of curare in the treatment of this condition.

At the present time the use of curare, while not actually in disrepute, is approached by many with trepidation. This is due not only to the sinister history of the "South American arrow-tip poison" but also to the general impression[6,9,13,14] that the drug does not lend itself to standardization and is a source of danger by reason of its variable, unpredictable, and at times catastrophic effect. Standard works on pharmacology make such statements as:

> "the possibility of its therapeutic use depends largely upon whether an adequate degree of muscular relaxation can be obtained without arrest of respiration, and therapeutic experiments have been hampered by difficulty in obtaining reliable preparations of curare or of its active principles. . . . It is not impossible . . . that if the active principles can be obtained and the dosage accurately determined, curare may yet find a place in therapeutics for the treatment of conditions of spasm and rigidity of voluntary muscles."[6]
>
> "Curare and its alkaloids have no valid, well-established clinical uses."[9]

Objection against the clinical use of curare on the basis of its variability was valid until the past few years. Despite this, the interest of many clinical investigators has been drawn to the possibility of its usefulness from the time of Jousset[11] who first utilized it therapeutically through the years.[15,17] The return of the Gill-Merrill expedition to the United States in 1938[8] with large quantities of curare made possible research on its standardization.

The importance of the availability of some means of control, other than surgical, of the mass reflexes resulting from cord transection, becomes obvious for definite reasons. The presence of mass reflexes in spastic states of the extremities as the result of cord injury is dependent upon the integrity of the spinal reflex arc. This condition can be adequately controlled by either an anterior or posterior rhizotomy. This procedure, however, must necessarily be reserved for those cases in which not even the slightest improvement can be anticipated, because the operation in itself is a disabling one. As far as is known, this leaves little to offer the patient suffering from mass reflexes in whom recovery of some degree is either taking place or is expected. A drug affording any relief from mass reflexes and spasticity is of definite therapeutic value.

The expansion of knowledge concerning curare is resulting in its widespread employment in anaesthesiology,[4,10] and in the prevention of the

traumatic effects of metrazol convulsions.[1,16] The successful utilization of curare in alleviating convulsions of tetanus has been reported.[5,17] Finally, the employment of curare in spastic states has been described.[2,3,7,12]

OBSERVATIONS ON THE USE OF CURARE

A preparation of curare has been employed in this hospital in the treatment of spasms of the lower extremities of 12 patients suffering with paraplegia secondary to transverse myelitis incurred as the result of wounds. Justification for the alleviation of spasticity is covered in the discussion and case reports of 5 patients most intensively treated. All the patients whose treatment with curare is described herein are young men with traumatic paraplegia in whom spastic and spasmodically contracting muscles of the lower extremities and trunk posed a threat against the possibility of either partial or complete usefulness. A total of 453 injections was given to these 5 patients up to 8 July 1945.

Dosage. 1. The drug was administered intramuscularly.

2. All of the 5 patients described in the appendix were given initial doses of between 0.020 and 0.100 grams, repeated not oftener than 72 hours.

3. In these same cases the amount of curare was increased by increments of (at first) 0.010 grams and (later) 0.020 grams until doses of 0.140 grams were reached.

4. Patients whose treatment we have initiated more recently have received "trial" doses of 0.060 grams, which were later modified to fit their needs.

5. The revised schedules stipulated doses varying from 0.050 to 0.070 grams, given at six-hour intervals.

Toxicity. 1. Mild toxic reactions were seen frequently. These were consistently seen when doses exceeding 0.080 to 0.100 grams were employed. When smaller but therapeutically effective doses were used, mild toxic reactions were observed in approximately 50 per cent of the cases. Severe toxic reactions were not noted. For a period of at least 60 minutes following the administration of curare, prostigmine and facilities for artificial respiration were kept in readiness for immediate use. This precaution has never been found to be necessary and after 3 weeks' experience with the drug, facilities for artificial respiration were not alerted. At no time was a change in pulse, blood pressure, respiratory rate or force observed.

2. The mild toxic reactions noted consisted of weakness in ocular convergence, droopiness of the lids and at times generalized muscular weakness. These were noticeably accentuated when the patient attempted vigorous physical exercise.

3. No cumulative effect was noted when the drug was administered 4, and in one case 5 times daily over a period of more than a month.

4. Diminution of the gag reflex or other cranial nerve weaknesses aside from the above were not observed.

Time of Appearance and Duration of Effect. 1. The drug effect became

manifest in from 15 to 25 minutes and reached a maximum in from 30 to 45 minutes.

2. With very few and questionable exceptions, the entire effect of the drug in this group of patients had disappeared in from 4 to 7 hours.

3. When curare is being employed for the purpose of facilitating the use of a walker or braces, it has been found best to administer the drug 1 hour before the patient begins this exercise.

Therapeutic Effect. 1. Spasticity was diminished to a noticeable degree in all patients for from 1 to 2 or more hours after every administration of an effective dose.

2. Spasticity returned gradually to its original severity in from 4 to 7 hours following injection.

3. In no instance did the effect of a single administration of curare persist for more than 24 hours.

DISCUSSION

Importance of Controlling Spasticity and Mass Reflexes. The control of spasticity in such patients as this paper describes is important for a number of reasons:

1. *Bed Sores.* Practically all patients, because of the nature of their wounds, the long distance over which they traveled, and related factors, were afflicted with extensive decubitus ulcers upon admission to this hospital. It is a universally accepted fact that the frequent turning of these patients (every 2 hours has been our infrangible rule) is of paramount importance in the cure and prevention of bed sores. The relief of spasticity by curare is important in this regard in facilitating the handling of these patients and in allowing them to be placed in positions that could not otherwise be assumed without being lashed down. The recent application of skin grafting as the final step in healing decubitus ulcers is not infrequently hazardous and certainly not an assured method because of the difficulty of immobilization of the grafted site. Curare has been efficacious in obviating this disadvantage.

2. *Aid to Physiotherapy.* Physiotherapeutic efforts to promote the return of muscles to more nearly normal tone are greatly impeded by the spasticity exhibited by these patients. This is often of such severity that any handling of the extremities initiates distressing mass reflexes. The use of braces and walkers by these patients frequently is impeded by extremities so spastic that they are a hindrance in getting about. The employment of curare has enabled spastic muscles to become facile enough to make the use of braces and walkers appreciably easier and more effective.

A graphic example of the effect of curare is that whereas before administration the patient's lower extremities have had to be strapped down with the thighs and legs in extension in order to prevent spasmodic flexion at the hip of such a forceful nature as to tend to throw patient out of bed, following curarization this strapping is no longer necessary (for varying intervals, as discussed).

Although no cumulative effect has been observed to date, it is reasonable to suppose, on the basis of experience, that when severely incapacitating spasticity is relieved for a long period, allowing physiotherapy to operate, and permitting the use of braces and walkers, partially functioning muscles will improve and parts completely out of control will at least not be so handicapping as before. The study of a large series, controlled adequately, will have to be protracted over several years to determine whether and how permanent a result is achieved.

3. *Rest.* It is not uncommon for these patients to be so annoyed by spasms as to be unable to sleep. The administration of curare at 7 P.M. or 9 P.M. and 1 A.M. or 2 A.M. was effective in diminishing the number and extent of the spasms. Several of our patients (notably Cases 1, 2 and 4) have volunteered that for the first time since the onset of their spasticity they have been able to enjoy an uninterrupted nightly repose. We have found that the size of these nightly doses could be increased over those given during the day. The toxic symptoms noted did not prove a source of annoyance during the sleeping hours.

4. *Diminution of Pain.* Since the action of curare is classically at the motor end plate, it would be expected that the diminution of pain would not be one of its important effects. Relief was observed in two cases. In both instances the pain occurred in a normally innervated region. Relief of pain was coincidental with the alleviation of the spasms effected by curare. It seems a reasonable assumption that the pain in these cases was due to the excessive pull of the spastic abdominal muscles on the sensorially intact tendinous insertions into the lower ribs.

Selective Action on Spastic Muscles. Our experience has confirmed that of Bremer[2] and Burman[3] that spastic muscles are affected by curarization with appreciably smaller doses than are normally innervated muscles. It was obvious after every injection that, whereas there was a notable decrease in the spasticity of the involved muscles, there was very little or no weakness produced in the normally innervated muscles. Some weakness was usually noticed in normal muscles when the patient exerted himself vigorously. This was, however, not comparable to the relaxing effect on the spastic muscles. We were thus enabled to achieve an appreciable decrease in spasticity and maintain a comfortable margin of safety in dosage. This margin was increased by the fact that we could attain as satisfactory relaxation of spasticity with moderate doses (0.070 to 0.100 grams). Larger doses (up to 0.140 grams) had a very annoying paralyzing action on normally innervated muscles but produced no greater degree of relaxation on spastic muscles.

Interval between Doses. The available literature does not mention the administration of curare oftener than once a day. This is not only apparently the rule in the use of the drug in anaesthesiology and as an adjuvant to metrazol convulsion therapy, but also in the treatment of spastic states.[3,7] The impression is given that the proper interval between doses is 2, 3 or 4 days. In fact, one author advises that "curare should not be repeated within 24 hours."[15] On this basis we originally administered curare to our patients

not oftener than once in 72 hours. It soon became obvious that this precaution was unnecessary. This over-cautious attitude was shown to be incorrect by administering curare to several patients 4 to 5 times daily over a period of more than a month. This was done with complete impunity. Furthermore, such frequent use of the drug is a therapeutic necessity in order to gain as complete an effect as possible.

Variability of Effect. The dose needed for optimal effect was not strictly correlatable with body weight. For example, Patient 1, whose weight is 10 to 15 pounds more than Patient 3, required 0.020 to 0.040 grams *less* for relief of spasticity. He likewise experiences annoyingly toxic symptoms at a dose level that is barely able to produce early drug effect in Patient 3.

There was a slight variation from time to time in the efficacy of the drug, as well as in its toxicity. This was minor in every case and easily managed.

Need for Slower Absorption. The fact that optimal effect could be achieved only by the administration of curare 4 or 5 times daily is not of grievous significance in a hospital where trained personnel and equipment are available. However, under other circumstances, and in the absence of trained personnel and equipment, the need for frequent dosage could be a severe handicap. Oral administration of the drug is not feasible. A preparation satisfying most needs would be one whose effect could be prolonged over a 24-hour period. This might be achieved by combining the curare with such a vehicle as beeswax. Such a preparation is being sought and efforts will be made to determine its efficacy.

CONCLUSION

1. The use of curare in the treatment of annoying and painful spasms of the lower extremities and abdominal muscles has been described and discussed.

2. Methods of administration previously unreported have been detailed and their use in 12 paraplegic patients has been delineated. Emphasis was made on the fact that the preparation of curare used in these observations may be injected 4 or 5 times a day over prolonged periods without harmful effect.

APPENDIX

Case 1—Age 24. Complete, spastic paraplegia, due to traumatic myelitis at the level of the 8th thoracic vertebra, following shell fragment wounds of the spine sustained in combat 22 January 1944. The patient was seen during a stage of his illness when spasmodic contractions of the lower extremities were immobilizing him to a dangerous degree (since any stimulus sufficed to bring about mass reflexes), but also were contributing to debilitation in that he was unable to get a night's rest on account of them. The spasticity of the lower extremities made it necessary for them to be kept firmly bound down in extension.

The patient was curarized; doses varied from 0.020 to 0.140 gm. He received a total of 62 injections up to 25 June 1945. The spasms were relieved from 10 to 60 per cent after various injections, the degree of relief not being strictly correlatable with the dosage or any other determinable factor. No abnormality in vital signs was ever noted, but when the dose exceeded 0.080 gm. the patient experienced rather annoying blurring of vision, a sensation of "droopiness" of the eyelids, and generalized muscular weakness upon vigorous exercise. All

of these symptoms passed off in about an hour after their onset. In 2 months' time after the beginning of curarization the tendency toward mass reflexes diminished greatly and treatment was discontinued.

Case 2—Age 33. Complete, spastic paraplegia, due to traumatic myelitis at the level of the 6th thoracic vertebra, following gunshot wounds of the spine sustained in combat 18 November 1944. Shortly after his injury the spasticity became marked and his lower extremities had to be tied down at all times, with the thighs and legs in extension. This was a factor in bringing about the onset and persistence of decubitus ulcers.

Accordingly, on 10 May 1945, the administration of curare was begun and has been continued up to the present time. He received a total of 92 intramuscular injections up to 2 July 1945. The amount has varied from 0.060 to 0.140 gm. No changes in vital signs were noted, and he has experienced excellent results as far as the relaxation of the spastic muscles is concerned. At times when the patient has been away from the hospital on pass he has noted therapeutic effect for about 24 hours, the maximum effect occurring from 1 to 4 hours following injection. Generally, the tendency toward spasticity has decreased considerably. His lower extremities no longer need to be tied down at all times, and his paralysis has passed into a stage wherein mass reflexes, while still occasionally present, are not so annoying.

Case 3—Age 22. Complete, spastic paraplegia, due to traumatic myelitis at the level of the 2nd thoracic vertebra, following gunshot wound of the spine sustained in combat 2 December 1944. Since February 1945, the patient had been annoyed by mass reflexes of the lower extremities which impeded the healing of bed sores and were extremely painful in that they made tense the subcostal tendinous insertions of muscles where sensory innervation was intact.

On 1 June 1945, the intramuscular administration of curare was begun. He received a total of 55 injections up to 9 July 1945, which varied in amount from 0.070 to 0.140 gm. A beneficial effect from each of the injections was noted. He now receives the drug 4 times daily, and the extremities need no longer be bound down during the major part of the day and night. This is true despite the fact that his tendency toward spasticity remains unchanged—during the last week in June, because of difficulty in ocular focusing which occurred after each injection and at the patient's request, the administration of curare was discontinued for a 10-day period. During this time the spasms returned with a force sufficient to make the patient ask to be curarized again, and to be maintained on curare, which is the case at present.

Case 4—Age 23. Complete, spastic paraplegia, due to traumatic myelitis at the level of the 8th thoracic vertebra, following a bullet wound of the spine sustained in combat 22 September 1944. Since his admission to this hospital in November 1944, he had been annoyed by mass reflexes of the lower extremities which occurred after minimal stimulation.

On 24 May 1945, the administration of curare was begun. The doses varied from 0.070 to 0.160 gm., and were followed in every instance by therapeutic effect, consisting in a 40 to 70 per cent diminution of the spasms. He received a total of 156 injections up to 8 July 1945. Curare has been given to this patient intramuscularly 5 times daily for more than a month without toxic effect other than transient convergence weakness and slight muscular weakness. This patient is one of those paraplegics who have become able to start using a walker. His use of the apparatus has been made easier by curare in that it markedly diminishes the awkwardness engendered by spasm of the thigh adductors.

Case 5—Age 30. Complete, spastic paraplegia, due to traumatic myelitis at the level of the 5th thoracic vertebra, following a gunshot wound of the spine sustained in combat 2 December 1944. At the time of his admission to this hospital in January 1945, his previously flaccid paralysis had become spastic. It was severe enough that his lower extremities had to be tied down in extension. He also complained of severe pain in the precordial region, brought on by mass reflexes which included spasms of the abdominal muscles. The pain was inter-

preted as being due to stretching of the tendinous insertions of the abdominal muscles in the area where his sensation was intact.

Accordingly, on 23 May 1945, curare administration was begun. He received doses of from 0.060 to 0.140 gm. intramuscularly, and was given a total of 88 injections up to 28 June 1945. At this time the spasticity, which existed following a period of a few days in which administration of the drug was stopped, was so mild that it was decided that his paralysis had entered a less spastic stage, and the curare was discontinued. During the period of administration, skin grafting was done on this patient's back. It was felt by the surgeons that in his and other cases the success of the skin grafting was made more certain by the diminution of spasmodic contractions of the lower extremities and trunk which was brought about through curarization.

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CASE REPORTS AND TECHNICAL NOTES

ANEURYSM OF THE INTERNAL CAROTID ARTERY ASSOCIATED WITH HYPOTHALAMIC FITS

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Aneurysms involving the circle of Willis are of common occurrence. In spite of their frequency, it is seldom that one attains unusual size, and it is a decidedly rare occasion when the history is found to extend over a period of 25 years. In Dandy's recently published work comprising 133 aneurysms, one case only covered a 22-year history, although there were several others in the age group of 10 to 17 years.[3]

The most unusual feature about the aneurysm described in the present report was not associated with its longevity or size, but rather concerned its influence on the hypothalamus. In Dandy's large series there was no case that in any way gave evidence of derangement of the functional activities of this center.[3]

CASE REPORT

A white 53-year-old veteran was admitted to the hospital on March 13, 1944. He complained of "severe headache over the right eye, trouble with his eyes, and fainting spells." The onset occurred 25 years previously in 1919 while serving with the armed forces in France. At that time he received a minor injury to his head, and, although dazed, was not knocked unconscious. He was able to recall a severe headache localized to the forehead and was of the opinion, but not certain, that he saw "double" with his left eye tending to turn out. At any event diplopia did occur, but whether instantly or over a period of time could not be stated definitely. Sometime later he experienced what he referred to as "fainting spells." These spells were characterized by a feeling of drowsiness which would rapidly increase in intensity, terminating in unconsciousness. There were no convulsive seizures associated with these episodes. The duration of the attacks varied from a few minutes to an hour or more.

FIG. 1. Photograph of the patient on admission, which demonstrates the cranial nerve involvement.

At the beginning, these "attacks" were not particularly frequent, probably averaging four or five a year. The progression of symptoms was gradual. The headaches became more severe; the "fainting spells" more frequent; and the left eye pulled further to the left. The vision, particularly of the left eye, became greatly impaired. For the six months previous to admission the headaches were unusually intense and frequently shifted to both temporal regions. The "fainting spells" were longer in duration and occurred at least once a day.

The other relevant history was that of a penile lesion acquired while in the Service. It was not a proven chancre, and antiluetic therapy was never given.

Examination. The patient was a rather poorly developed and poorly nourished chronically ill white male. He was rational and oriented, but exhibited some evidence of memory impairment. His gait was normal, but he walked with obvious fatigue and his head was tilted and fixed to the right.

Neurological examination showed the following positive findings: There was atrophy of the left disc, but fingers could be counted at several feet. The right disc was slightly blurred. The left 3rd and 4th cranial nerves were paralyzed (Fig. 1). There was complete sensory and partial motor impairment of the third division of the left 5th cranial nerve as well as a right central facial weakness. The peripheral reflexes were present and equal throughout, but on the hyperactive side. There were no pathological ones present. The Romberg test was negative. There was some clumsiness of the left arm, and left past pointing was fairly marked.

Laboratory tests, including the serology, were within the normal range. Spinal fluid examination was not done.

The x-ray report of the skull stated that there was an extensive calcification in a cyst wall occurring above and posterior to the sella, displacing the tip of the dorsum downward and forward, and situated chiefly to the left of the midline. Three distinct lobes of the cyst could

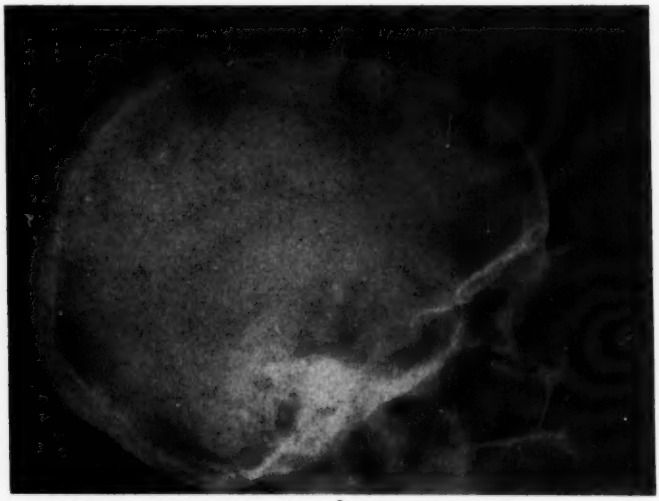

FIG. 2. Lateral film, showing marked calcification in suprasellar region. The curved linear lines supposedly characteristic of an aneurysm are present. There is some demineralization of the sella, particularly noticeable in both the anterior and posterior clinoid processes. The latter are tilted forward and depressed. The floor of the sella is not distorted. Actually there is minimal change in the sella for a tumor of this type and size.

be seen. The total width of the tumor mass was almost 6 cm. (Figs. 2 and 3). The x-ray opinion was craniopharyngioma.

Course. The patient's hospital course was marked by increasing severity of headache and "fainting spells." On observation these "spells" were characterized by an initial feeling of weakness and faintness. These aura were usually of sufficient length to enable him to reach his bed before unconsciousness intervened. This was accomplished without attracting attention, and other patients on the ward were totally unaware of the impending attack. He would simply climb on his bed, lie down quietly and, after carefully supporting his head, slip into deep unconsciousness. There was no evidence of motor involvement. Rapidly the color would become pale; the skin cold and clammy; the pulse weak and, at times, almost imperceptible. The blood pressure always fell from an average of 130/70 to 100 systolic or below, and on several instances it was necessary to give the patient supportive intravenous therapy.

At the termination of these periods of unconsciousness, the patient was frequently, but not always, confused. Within a few moments complete orientation returned and the only evident effect was the ever present headache.

Operation I. On March 22, 1944 a left frontal craniotomy was performed and, in the parasellar region, a solid tumor mass was encountered. It was tapped with difficulty with a fine needle but no cystic areas were disclosed. The chiasm was markedly taut and stretched.

Course. Postoperatively, the patient stated that he felt improved; whereas formerly he had had several "fainting spells" daily, he now remained free until April 1, 1944, when it was noticed by the ward nurse upon bathing him that he suddenly could not be aroused. His hands and feet became cold; his pulse thready and weak; respirations shallow; and his color pale. In approximately an hour, he began to respond. His temperature was reported as 96.2; pulse 70, but still weak.

During the next 3 weeks, there was definite general improvement; the headaches were less severe. He requested leave and on April 25, 1944 was allowed to go home for a few days.

FIG. 3. Posterior-anterior view, which shows the extensive character of the aneurysm. It measured 6 cm. in width. The photograph depicts well the left lobe of the tumor but has not reproduced the upper or right lateral lobes.

He was readmitted May 12, 1944. The temperature was 98; pulse 80; respirations 16. He complained of increasingly severe headache and "fainting spells." His condition progressed and on May 24 he became drowsy. For the first time he vomited. There was clinical evidence of increasing intracranial pressure. During this period his blood pressure averaged 102/84.

Operation II. On May 25, 1944 the bone flap was elevated and a portion of the frontal lobe was removed for decompressive purposes. Further exploration of the tumor failed to reveal any cystic areas.

Course. A superficial wound infection became evident on the 5th day postoperatively. Meningitis followed associated with left-sided convulsions, and 10 days later he expired.

Necropsy. A large tumor mass, measuring $5.5 \times 4 \times 2$ cm., was present in the middle cranial fossa. The tumor was extra-cerebral, but deeply notched the hippocampus and uncus gyri of the left temporal lobe, and distorted all structures extending from the pons rostrally.

The cranial nerves from the 2nd to the 5th bilaterally were difficult to identify. The left 5th was incorporated in the capsule of the tumor mass as were the 3rd, 4th, and 6th nerves on the left. The basilar vessels were greatly changed with respect to anatomical relationship due to distortion by the tumor mass. Before removing the tumor, it was noted that the left internal carotid artery appeared to pass through its central portion. The optic chiasm was deformed, and the optic tract was thinned on the left. The tumor itself was solid, greyish-brown in color, encapsulated, and the external surface was irregularly nodular. There was moderate erosion of the left anterior clinoid process with involvement of the dorsum of the sella, and depression of both posterior clinoid processes. The pituitary gland was normal in size and appearance as was the floor of the sella.

Upon removal of the tumor, a large crater was exposed. It was bounded by the temporal

FIG. 4. Aneurysm of left internal carotid artery shown in exact relationship to the circle of Willis. The right posterior communicating artery is seen as a group of thread-like structures which dwindle to a single strand. The left posterior communicating artery could not be identified. The left posterior cerebral and superior cerebellar arteries are definitely larger than those of the right side, thus compensating for the absence of the left posterior communicating artery. Similarly, the anterior cerebral and the medial artery of the corpus callosum are increased in size.

lobes laterally, and all involved tissues were moulded and flattened against the sides. The floor of the depression was concave, and the hypothalamic region was compressed and distorted with the exception of the mammillary bodies which, although shifted backward in position, retained their normal gross form. The left cerebral peduncle was extensively gouged. The tuber cinereum and infundibulum were so smoothed out that it was difficult to distinguish them from the under-surface of the chiasm. The circle of Willis was unusual in that the left posterior communicating artery could not be identified and the right was thread-like in size. The left posterior cerebral artery was increased in size over that of the right side, although both were larger than normal. The circle of Willis was removed and its relationship with the tumor mass is shown in Fig. 4. On sectioning the tumor, concentric laminations of partially calcified thrombi of various ages were disclosed. The mass was solid, but some evidence of late hemorrhage in the form of a recent thrombus at the entrance to the sacculation was present.

The aneurysm came off the left internal carotid artery at a point where I would judge the left posterior communicating artery should arise. The opening was small and comparable with that of a normal posterior communicating artery. The lumen of the internal carotid was completely patent with no evidence of occlusion or obstruction throughout its entire length. Anatomically it was shown that adequate circulation through the circle of Willis had been maintained.

DISCUSSION

This case is noteworthy not only from the point of view of the aneurysm itself, but also from the clinical picture of hypothalamic involvement.[5,6,8] In the first place, the fact that an aneurysm of such large size could have presented so few manifestations generally attributed to aneurysms of this location during its 25-year course seems quite remarkable.[3] Undoubtedly, the many episodes of bleeding, as evidenced by the calcified laminations, finally resulting in spontaneous cure, as shown at postmortem examination, were caused by leakage rather than rupture. Certainly, there was no incident in the history compatible with or suggestive of a major rupture. Also the pulsatile characteristic must have terminated early or have been confined to a minimum; otherwise, more destructive changes in and about the sella would have occurred. Comparably, the amount of change in the sella was slight. The left anterior

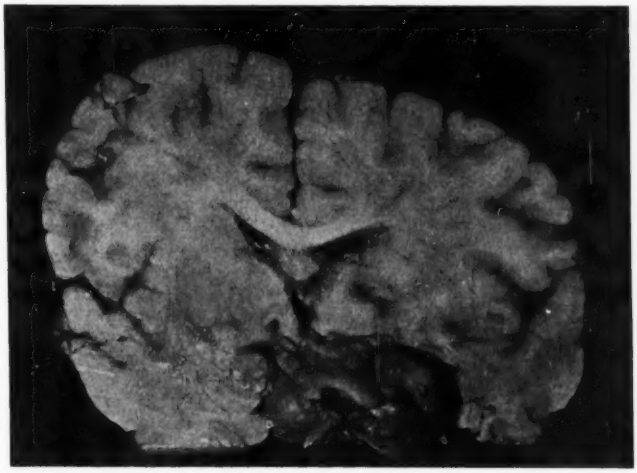

FIG. 5. Coronal section, which shows the aneurysm *in situ*. A stick is placed in the left internal carotid artery, which was found to be completely patent. Marked distortion and flattening of the chiasm and hypothalamic region are evident. The ventricular system, with the exception of slight enlargement of the third ventricle, is within normal limits.

clinoid process was eroded and some general demineralization was noted throughout. These abnormalities together with forward bending of the posterior clinoid processes comprised the total structural change. The floor of the pituitary fossa was smooth and normal in shape.[3]

The second unusual feature was the lack of development of obvious increased intracranial pressure associated with a lesion of this location and size until late in its course.[2] A coronal section of the brain suggested a slight enlargement of the third ventricle with normal-sized lateral ventricles, as shown in Fig. 5.

From the clinical point of view, the most interesting feature was that described by the patient as his "fainting spells." The first time he was observed in one of these spells, his picture seemed indistinguishable from that of surgical shock. The color was poor; the body cold and clammy; the temperature subnormal; respirations shallow; the pulse weak and thready; and the systolic pressure below 100. On several other occasions, as mentioned above, intravenous support was given during these periods followed by definite improvement in his general condition. If left untreated, gradually he became conscious in conjunction with the return of his color, pulse, and blood pressure to the former levels.

Preoperatively, his mouth temperature consistently ranged between 97.2 and 97.4. The

day following his first operation, the temperature rose to 99.2, and then varied from 96.4 to 98 with a pulse averaging 70 and respirations 20. Only on the day of discharge did the temperature reach 98.6. There was this evidence of continuous disturbance of the heat-regulating center which became more pronounced during the periods of the hypothalamic fits.[2,4,8] There was no clinical evidence of any anterior hypothalamic irritation or destruction. Although the tuber cinereum was crushed and thinned, functionally it remained intact and there was not the least indication of disturbance of fat, carbohydrate, or water metabolism. There were no emotional crises nor was there any evidence of mental instability. The patient's emotions were always under control, and although his general picture was one of dullness and sleepiness, this was not borne out by his mental response.[1]

The question is raised by these episodes, which might be classified as a type of autonomic epilepsy, as to whether there is not a distinct relationship between surgical shock and elimination by depression alone of the posterior hypothalamic nuclei.[7] It is conceivable that a vast flow of impulses instigated by trauma could be a sufficient reason for fatiguing these nuclei and thereby initiate the rapid development of shock.

CONCLUSION

The clinical and anatomical record of a large sacculated aneurysm of the internal carotid artery of 25 years' duration has been presented. The clinical manifestations of associated hypothalamic fits have been discussed. The intimate relationship of the posterior hypothalamic nuclei and surgical shock has been suggested.

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PINEAL TERATOMA: REPORT OF CASE

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Tumors of the pineal body remain puzzling lesions from the standpoint of their origin, their true nature and proper designation, their potentialities for endocrine disturbance and their treatment. Teratomas have been invested with especial interest principally because of

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the unsolved problem of the relation of this type of tumor to the syndrome of macrogenitosomia praecox. Bochner and Scarff[4] have provided a recent review of the literature of these particular tumors in reporting a case of their own. They found 13 tridermal and 4 bidermal tumors and, in addition, 10 tumors that they considered to be compound or mixed tumors similar to certain tumors of the salivary glands. Since the mode of origin of all these tumors is beclouded and since Hosoi's[13] classification of certain of these tumors conflicts with Bochner and Scarff's, the latters' distinctions with regard to certain of these tumors may be artificial and McLean's[15] larger collection of teratomas may come closer to representing the true total of tumors of this particular type. McLean found 26 tumors that he considered to be teratomas. He did not include one case of teratoma and one case of teratoid mentioned by Bochner and Scarff. Zeitlin's[23] teratoma (case 1) seems to have escaped Bochner and Scarff and should be included, as should the case reported by Ragan.[19] Certain additional cases have been reported recently. Baggenstoss and Love[2] in their case 4 and Russell[20] in her case 2 reported what may be other examples. The Quarterly Cumulative Index Medicus contains references to certain other reports which were not available. Thus, it seems that more than 33 termatomas, teratoids and mixed tumors have been observed.

A patient who had a pineal neoplasm was recently under my care at the Mayo Clinic.

REPORT OF CASE

The patient, a youth aged 18 years, registered at the Mayo Clinic on March 28, 1944. Because of the nature of his complaints he was hospitalized immediately. His reason for coming to the clinic was headache. He stated that on Feb. 15,1944, he began to have intermittent bilateral frontal headaches which at first lasted for 2–3 days and which became progressively more severe. Three weeks prior to registration, the headaches became constant and were made worse by sudden movement of the head and by straining. The headache never awakened him but it often prevented him from going to sleep. Some time after the headaches began, he noted giddiness and nausea and soon after the onset of these symptoms he began to vomit intermittently. He was hospitalized elsewhere and spinal puncture was done which revealed an increase in the intraspinal pressure. For a few weeks prior to registration at the clinic, he experienced weakness and intermittent numbness in the left leg and more recently there had been some vague diminution in visual acuity.

His left eye had been removed after an injury at the age of 4 years. Both parents were living and well, as were eight brothers and one sister. The patient had had no previous significant diseases.

Examination. He was a well-developed and well-nourished young man with the significant findings limited to the nervous system. Examination of the cranial nerves revealed no defect except for inability to elevate the remaining eye more than slightly above the horizontal. The patient stated that attempts to elevate it further caused pain in the orbit. The visual acuity of the eye was 14/21; the pupil was large and reacted neither to light nor in accommodation. A rough examination of the visual fields revealed no defect. Examination of the ocular fundus showed acute choking of the disk amounting to an elevation of 3 diopters. There were hemorrhages around the disk and some small ones beyond the macula. The veins were moderately engorged. The muscles of the left calf were slightly weakened but all other muscles possessed normal strength and tone. All tendon reflexes were equal and of normal intensity. There was no disturbance in sensation nor defect in coordination or in gait.

Routine examinations of blood and urine gave negative results. Roentgenograms of the head disclosed a considerable amount of calcium lying in the region of the pineal body. An electro-encephalogram disclosed bilateral delta activity in the occipital regions which was interpreted as being due to a lesion in the posterior fossa.

On April 1, 1944, the patient had persistent vomiting with twitching and clonic movements of both legs. The fingers of the right hand became numb but feeling returned intermittently. Diagnoses entertained at one time or another by those observing the patient were: right parasagittal lesion in the central area, lesion of the posterior fossa, and lesion of the midbrain.

Ventriculography was carried out April 3, 1944, through bilateral holes placed in the posterior part of the parietal region. The ventricle on each side was entered at a more shallow depth than is normal and 160 cc. of fluid was obtained. However, the fluid did not appear to be under increased pressure. The ventriculograms (Fig. 1) revealed the presence of a tumor at the posterosuperior aspect of the third ventricle which was considered to be a pinealoma. A soft rubber catheter was left in the right lateral ventricle and the patient was returned to his room.

Operation. On April 7, 1944, after the ventricular drainage had been attended by improvement in the patient's condition, right parieto-occipital osteoplastic craniotomy was performed. The opening was of moderate size.

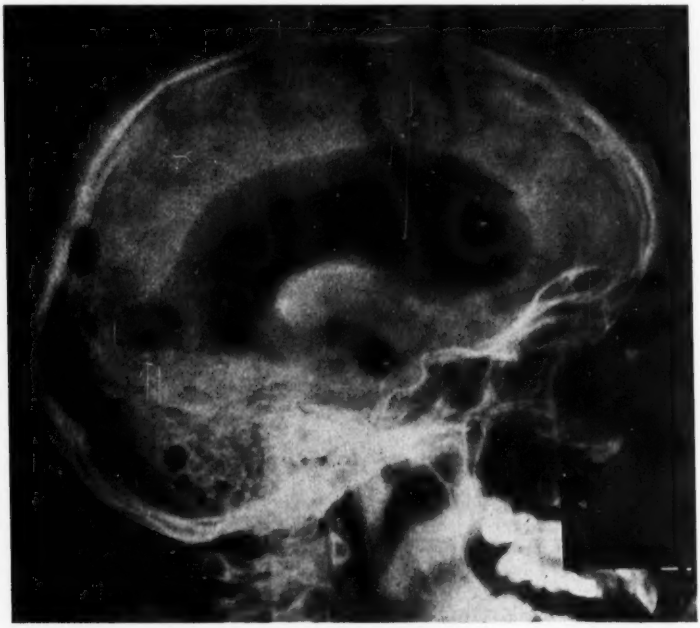

FIG. 1. Lateral ventriculogram in a case of pineal teratoma. The tumor is well displayed as an irregularly spherical mass at the posterosuperior aspect of the third ventricle. The superior aspect of the tumor lies on the same horizontal level as the upper border of the third ventricle. Note the delineation of the angle between the curving anterosuperior border of the tumor and the roof of the ventricle.

Bone was rongeured away to the midline until the superior sagittal sinus was exposed for a distance of 3 inches (7.6 cm.). A large dural flap was reflected medially. A single large vein extended from the posterior parietal region to the superior sagittal sinus. This was divided between ligatures. The right lateral ventricle was punctured and fluid withdrawn as the brain was retracted laterally away from the falx. The splenium of the corpus callosum was divided for a distance of 1 inch (2.5 cm.) and a nodular tumor was immediately apparent. It lay beneath the veil of connective tissue in the transverse cerebral fissure. Because the lower border of the falx prevented exposure of the whole mass, it was necessary to divide the falx. A number of venous channels in the falx bled sharply and it was necessary to use a number of silver clips. The divided margins of the splenium then were retracted to each side and the connective tissue and the internal cerebral veins were teased off the surface of the tumor.

The left internal cerebral vein was torn in doing so and the bleeding point was gently packed off with a strip of cottonoid. The tumor was grasped and gently rocked from its bed. It had no firm attachment to underlying structures. As the tumor was removed, a moderate amount of ventricular fluid welled into the wound from the anterior portion of the tumor bed but the aperture into the third ventricle was not looked for or into. A pledget of muscle was placed over the bleeding point in the left internal cerebral vein and when bleeding was controlled the tumor bed was perfectly dry. The dura was closed and the bone flap wired in place with two steel wire ligatures. Three grams of sulfanilamide was placed in the wound superficial to the dura and bone, since the catheter had been in place three days. Throughout the operation the patient remained in excellent condition. Nevertheless a transfusion of 500 cc. of citrated blood was begun as closure was being completed.

Course. The patient was returned to his bed and placed in an oxygen tent for 24 hours. He recovered from the anesthesia rapidly and soon was responding normally. The evening of the operation the temperature rose to 101°F. and thereafter rose to around 100° each afternoon until the 9th day after operation, when it became normal and stayed so. On April 24, 1944, he was dismissed with the wound well healed and in excellent general condition. The eye had begun to show more strength on upward gaze and although the pupil was still large there was some reaction in accommodation and to light. The remainder of the neurologic examination, including rough visual fields, was negative except for persistent papilledema.

On Aug. 22, 1944, the patient returned at my request for re-examination. The pupil was still large and measured 6 mm. in diameter and its range of contraction to light was small; it reacted normally in accommodation. The optic disk was structurally full, but there was no edema. When the patient looked far to each side, a few small nystagmoid jerks were apparent. The visual fields, accurately plotted, were normal, both to the 1 mm. object at a meter's distance and the 3 mm. object at 33 cm. distance. Visual acuity was 14/14. The remainder of the neurologic examination was negative. Roentgenograms of the head revealed that the bone flap was in good condition and the calcification previously noted in the region of the pineal body was absent.

On Nov. 30, 1944, the patient returned to the clinic stating that about the middle of September, 1944, he began to experience mild bitemporal headaches every ten days or so. These headaches were nonthrobbing, felt like pressure from within, were brought on by physical exertion, did not awaken him at night, were unaffected by change of position of the head and lasted for about a day at a time. They gradually increased in frequency until they were present every other day. On Nov. 1, 1944, he began to vomit and did so 3–4 times a day for 4–5 days when the vomiting suddenly ceased.

On Nov. 15, 1944, the vision in the eye became less acute and got steadily worse. He complained that everything appeared dark and that colors were particularly difficult to distinguish. The relative who accompanied him stated that he seemed to lack pep since the middle of October, 1944, that his memory was poor for recent events and that he seemed drowsy and sleepy all the time. During the two weeks prior to this registration he had complained of generalized sensations of pins and needles.

The pertinent findings on examination were nystagmus on looking to the right, visual acuity in the right eye of 3/60, with correction 14/42, almost normal reaction of the pupil in accommodation but only slight response to light, complete loss of vision in the nasal half of the field of the right eye to all sized objects, slurring speech, slightly diminished corneal reflex, awkwardness in tapping the toes of either foot on the floor and inequality of the patellar and Achilles reflexes. The reflexes on the right side were slightly more active than on the left.

Choking of the optic disk and limitation of motion of the eye in any direction were not present. Ability to hear a watch tick was recorded as 6/10 on the right and 10/10 on the left side. No audiometric testing was done. Superficial and plantar reflexes, co-ordination, gait, station and all forms of sensation were normal. Routine laboratory studies gave results within normal limits.

A ventriculogram was made on Dec. 12, 1944 by Dr. Kloos. Again the fluid did not seem to be under increased pressure. Dilatation of the lateral ventricles revealed little air in the

third ventricle but it appeared to contain a large mass. The lateral ventricles were of smaller size at this examination than they had been formerly. The fluid contained 20 mg. of protein per 100 cc., no globulin and one lymphocyte per c.mm. The ventriculography made it apparent that the recurrence of symptoms was due to recurrence of the tumor and not to postoperative stenosis of the aqueduct. Four roentgen treatments, one through each of four portals, were given. The factors were 200 kv., 50 cm., 20 ma. for 20 minutes filtered through ¾ mm. of copper and 1 mm. of aluminum. The patient was then allowed to return home.

A letter received from the family stated that he grew steadily weaker and died Jan. 16, 1945, without experiencing any more headaches after his return home. Necropsy was not performed.

FIG. 2. The pineal teratoma removed at operation.

Pathologic Findings. The tumor (Fig. 2) was a roughly spherical encapsulated mass measuring about 3.5 cm. in diameter and weighing 10 gm. Its generally spherical aspect was distorted by superficially located cysts of all sizes up to 1 cm. in diameter which made the surface grossly nodular. Some of these cysts had thin translucent walls while others had thicker yellowish ones. A few veins coursed over the surface at random. No broken tissue area was discernible to indicate its point of attachment. The cut surface displayed its polycystic nature well. Certain of the cysts contained cloudy fluid whereas others contained clear fluid.

Histologic preparations revealed the presence of numerous small cystic spaces not visible to the unaided eye together with islands of solid tissue bearing a number of different structures. The general nature of the tumor as seen in the sections is shown in Fig. 3. Among the structures present in the tumor were islands of embryonic cartilage. Fig. 4*a* shows the character of the walls lining some of the cystic spaces. In addition to what are properly called cystic spaces, were other smaller structures amounting to no more than an alveolar arrangement of cells in the fibrous matrix. Some of these are apparent in Fig. 3.

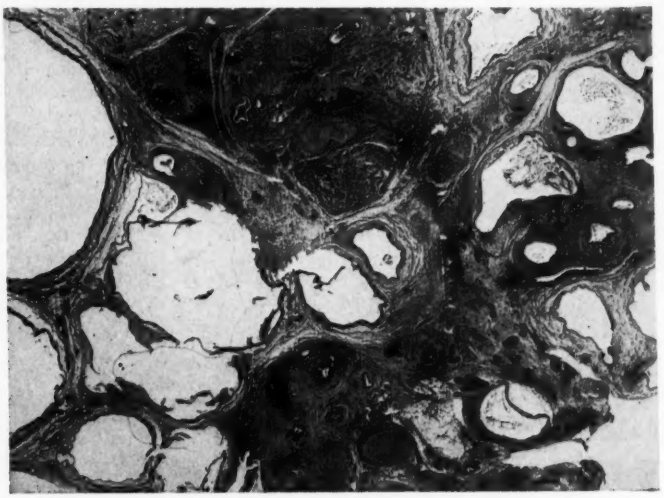

FIG. 3. General character of the tumor (hematoxylin and eosin, ×7).

But all these are of minor interest compared to what was seen in only one small area of the tumor; namely, poorly defined cords and masses of large oval and polyhedral cells interspersed with numbers of smaller cells with small dark-staining nuclei. Fig. 4*b* shows this region. Its similarity to the appearance of the tissue composing the tumor known as pinealoblastoma is evident. This area was found in the interior of the tumor.

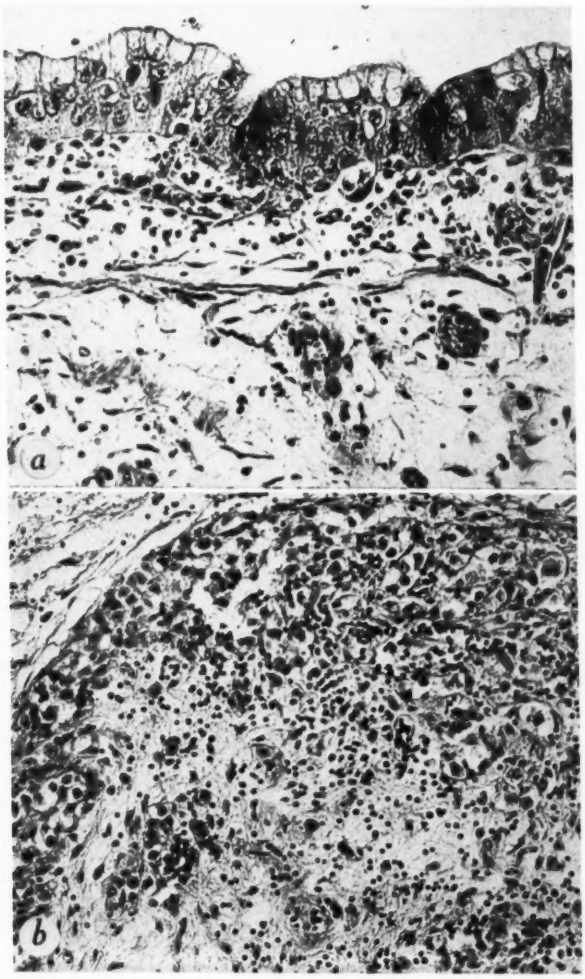

FIG. 4. *a*, Mucus-secreting epithelium lining a cyst (hematoxylin and eosin, ×200); *b*, the single small area of the tumor that bears a resemblance to pinealoblastoma (hematoxylin and eosin, ×140).

DIAGNOSIS

The symptoms and signs produced by pineal tumors arise through obstruction of the exit of ventricular fluid from the posterior end of the third ventricle, through paralysis of con-

tiguous nuclear masses and fiber tracts by the pressure of the enlarging mass and, possibly, by the formation of endocrine substance in the tumor itself.

The pro and con of formation of endocrine substance by the pineal neoplasm and of interference with supposed pineal secretion by the nonfunctioning neoplasm and the causation of the Pellizzi syndrome has been argued to no decision by others and the case reported herein adds no information. Weinberger and Grant's[22] recent hypothesis that the Pellizzi syndrome is due to contiguous pressure of nonfunctioning tumor on pathways controlling function of the anterior pituitary, if substantiated, would place the sexual and somatic aberrations in the same class as the ocular palsies and cerebellar signs as far as genesis by the pineal mass is concerned.

The symptoms produced by obstructive hydrocephalus almost invariably initiate the clinical picture, bring the patient to medical attention and are generally the ones most prominently displayed by the patient. Such was the case with the patient reported herein. This group of symptoms and signs produced by the tumor because of its relation to certain mesencephalic structures make possible clinical differentiation from other intracranial tumors. Paralysis of conjugate movement of the eyes upward, a central type of deafness and internal ophthalmoparesis are the three findings of most importance. The paralysis of upward gaze has received particular attention and is generally considered suggestive of pineal tumor. Harris and Cairns[9] and Bailey[3] warn that other tumors, particularly in children, may produce such weakness and others of experience have made the same observation. It hardly needs mentioning that not all pineal tumors produce this phenomenon. A variety of ocular abnormalities of a motor nature, anisocoria, failure of the pupils to react to light, ptosis and paralysis of extra-ocular muscles, including faulty convergence, attend or sometimes substitute for the paralysis of upward gaze in cases of pineal tumor.

The central type of deafness with predominant loss in the higher ranges may, on occasion, be unilateral according to Harris and Cairns.[9] The pathologic toe and extremity reflexes and facial paresis appear to be due to a combination of hydrocephalus and direct pressure on the motor pathways in the mesencephalon. The tendency to fall backward and the cerebellar signs seen in some cases are explicable on the basis of direct pressure, probably on the brachium conjunctivum.

TREATMENT

Tumors of the pineal body may be handled in either one of two different ways. Horrax and Daniels[11] have stated the case for the use of the conservative regime and few would disagree with their general proposal that in most instances pineal tumor is best treated by this means until an attempt at removal becomes mandatory. This state of affairs is true only because of the extreme hazard attending operative removal of the tumor and not because of any permanent cures resulting from the use of decompression and roentgen rays. Dandy[6] since 1921 has maintained that attempted operative removal is the only treatment worth considering. Kahn's[14] view regarding the preferred form of treatment is somewhere between that of Horrax and that of Dandy. He advocated attempting removal of all tumors that appear circumscribed in the ventriculograms, indicated by the preservation of the suprapineal recess. If the tumor upon exposure is found to be infiltrating, he advised performing a decompression and irradiating the neoplasm without even taking a biopsy. Dandy, incidentally, disputed the existence of a suprapineal recess in cases of pineal tumor. In certain cases, including the one reported herein, however, the ventriculogram reveals a recess above the tumor, but perhaps this is not the suprapineal recess of normal anatomy and should be termed "suprapinealoma recess."

If the adequacy of a procedure is measured by days that the patient survives, it is probable that conservative treatment is the one of choice. On the other hand, if the adequacy of treatment is measured by the chance it offers of producing permanent relief, then radical extirpation of the tumor is the procedure of choice.

The historical aspects of pineal surgery have been considered by McLean[15] and it is not profitable to review all the various measures suggested at this time. It appears, however, that

Horsley[12] was the first to record, fragmentary as the record is, attempts to remove a pineal tumor. He stated in 1910 that he had been using the infratentorial approach but that in the future proposed to approach the tumor from above and split the tentorium. Such an approach was used by Brunner[5] in 1913. The unilateral transoccipital route with section of the splenium of the corpus callosum and the tentorium is known by his name. Horrax[10] modified this approach to include resection of the occipital lobe in cases of large tumors not adequately exposed without this resection. The unilateral transparietal approach was described in 1921 by Dandy[6] and has proved its usefulness in a host of interventions in this region. Dandy has found it necessary to resect the occipital lobe in a few cases in order to gain room. The transventricular approach of Van Wagenen[21] is apparently useful in special cases in which the tumor is shown to project into the medial side of one lateral ventricle. Baggenstoss and Love[2] found it necessary to remove a portion of one tumor by Dandy's operation and the remainder by the infratentorial route three days later. As Baggenstoss and Love remarked, no single approach is suitable for all cases. Dandy's, however, appears to be in most favor. It provides ample room for most removals and in the exceptional case the occipital lobe may be resected and the incisura of the tentorium enlarged to gain more room. This approach is attended by the least danger of damage to important structures. The case reported in this paper demonstrates that removal of a tumor can be accomplished by this method without leaving permanent residues. Naffziger[16] and McLean[15] both remarked on the value of internal decompression by section of the tentorium in cases in which irremovable tumors are impacted beneath it or in its incisura.

Successful surgical removal of a pineal neoplasm, by which is meant removal of all or practically all of a tumor without death at operation or in the immediate postoperative period and disregarding the postoperative condition of the patient, has been reported for perhaps a dozen or so patients. The following surgeons, at least, have accomplished this deed: Van Wagenen,[21] Harris and Cairns,[9] Peet[17] (twice), Dandy[7] (thrice), Horrax,[10] German,[8] Kahn[14] and Pratt and Brooks.[18] In numerous other instances tumors of origins other than pineal have been removed successfully from this same intrinsically difficult location. The number of cures accruing from the removals reported is exceedingly small. In 1937 Kahn[14] recognized only two, Peet's and his own and at that time only one year had elapsed since the operation in his case. To this total of one case of definite cure and one of hopeful cure should be added that of Pratt and Brooks[18] who reported the case of a patient alive and well $3\frac{1}{2}$ years following removal of the tumor.

The case being reported in this communication is of some interest because of the lack of postoperative neurologic residues. Of all the cases mentioned in this paper only three patients escaped postoperative homonymous hemianopsia (Peet, Dandy, and Pratt and Brooks). Dandy[7] reported that his patient had constricted fields but no hemianopsia. Horrax'[10] doubt of whether hemianopsia could ever be avoided after extirpation of a pineal tumor is unfounded but the danger and even likelihood of its development remain nonetheless, due perhaps to occlusion of the internal occipital vein mentioned by Harris and Cairns.[9] In the case reported herein this vein was not seen and, if present, evidently was not damaged. Pratt and Brooks'[18] observation that their patient's pupillary reactions to light returned the evening of surgery and the uniformly spectacular nature of her whole recovery are worthy of note.

PATHOLOGY

Heretofore the pineal body has been considered to have potentialities for forming three general types of tumors: teratomas and teratoids, pinealoblastomas and rather uncommon tumors of presumed glial origin. Recently Russell[20] has drawn attention to certain relationships between the teratomas and the pinealoblastomas and has stated that the pinealoblastoma or pinealoma is, in reality, a teratomatous tumor. One of her arguments was that teratomas often bear regions of pinealoblastoma in them. The case reported in this paper seems to substantiate this idea. A predominantly cystic and benign-appearing encapsulated tumor containing a small region of pinealoblastoma recurred in a short time following what appeared to be total extirpation.

SUMMARY AND CONCLUSIONS

Pineal tumors are most difficult lesions with which to deal but at the same time are of great interest and extend many invitations for investigation and elucidation. Pinealoblastoma, teratoma and glial tumors comprise the growths arising from the pineal body and in that order of frequency. In the case reported a temporarily splendid result occurred after what was thought to be complete removal of a teratoma, but rapid recurrence followed. This case gives support to the recently advanced idea that pineal teratomas are capable of displaying histologic pictures of considerable variety all the way from benign-appearing cystic masses, perhaps containing teeth, to obviously malignant highly cellular tumors.

The operation of Dandy was utilized in the case reported and the postoperative freedom from hemianopsia or other deficiency indicates that removal of a pineal tumor through this approach is not invariably followed by residues due to operative trauma.[1]

The outlook for permanent cure even with seemingly total extirpation is not bright. Perhaps three such recoveries are on record but at the time of the reports not all of the three patients were past the period in which recurrence might be expected.

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TECHNIQUE OF PANTOPAQUE MYELOGRAPHY

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Pantopaque myelography is basically a simple procedure, consisting of the injection of contrast material into the subarachnoid space, fluoroscopy with spot films of the indicated levels, and removal of the contrast medium. Those who have had experience with the procedure, however, will agree that it involves many pitfalls and possibilities of error, all of which, it is believed, have been experienced by the authors. These misfortunes include bloody taps, formation of possible hematomata or collections of cerebrospinal fluid in the epidural or subdural spaces, elicitation of root pain, injection into the subdural instead of the subarachnoid space, failure to obtain films of diagnostic quality, failure to include landmarks for indentification of levels on the films, difficulty or failure in removing the contrast medium, and in cervical myelography, spilling of the contrast medium into the basal cisterns.

A constant attempt has been made to reduce the incidence of failures and to increase the speed and accuracy of the examination. The procedures outlined below represent a compilation of the written and personal communications of Hampton,[5,6] Kubik,[6] and Spurling,[11] various articles on the subject,[1,3,4,8,9,10] and the experiences of the authors and the other medical officers who have done over 675 myelographic examinations with pantopaque at the Walter Reed General Hospital.

The general technique of myelography is well known and is in daily use in most clinics. The purpose of this paper, however, is to stress a number of details that the authors and coworkers have found to markedly reduce the difficulties mentioned above.

EQUIPMENT USED AND PREPARATION OF PATIENT

An autoclaved myelogram tray is available for each patient. This includes the usual setup for a lumbar puncture except that a short bevel 3-inch 18-gauge Luer-Lok needle with stylet is used. In most instances the patients are able to walk to the X-Ray department; no premedication is used, and no meals are withheld. In fact, the procedure is given only casual consideration in the presence of the patient. Clothes are removed to the waist and the trousers are dropped down over the hips. The patient gets on the table and lies prone, with a folded pillow under the lower abdomen and pelvis to eliminate partially the lumbar curve and widen the spaces between the spinous processes. The prone position has been found preferable to the lateral position for two reasons: First, because it is easier to place the needle directly in the midline, and secondly, because it avoids possible changes in the depth of the needle point brought about by alterations of the skin-muscle-bone relationships attendant on turning the patient for X-Ray examination. The skin is then prepared, with gauze on a sponge stick, by vigorous scrubbing with some colored antiseptic solution, from about L-1 to the sacrum with about 3- or 4-inch margins on each side. A sterile towel is then placed over the trousers and upper buttocks. No other drapes are used because of their tendency to slide when tilting the table. Extensive drapes contaminate the field, give a false sense of security, and also obliterate the outline of the back, thus obscuring the spine for accurate alignment of the needle. The head of the table is tilted up about 25 degrees in order to increase the pressure in the lumbar sac. This gives more resistance to the needle on the part of the dura and arachnoid and permits more nearly perfect penetration of the needle through these membranes, thereby diminishing the chances that a portion of the bevel of the needle may rest in the subdural space.

The spine is then palpated with gauze and the spine of the 3rd lumbar vertebra is identified. The interspace below this spine is selected to avoid having the hub of the needle obscure the 4th or 5th interspace, at which, in our experience, practically all of the ruptured discs

have occurred. By the same token, any defect created by the needle point will be removed from the site of likely pathology and the chances of the needle striking a protruded disc are minimized.

A small amount of some local anesthetic has proven adequate. A small wheal is made over the interspace and not more than ½ cc. is injected into the subcutaneous tissues since greater amounts have been found to obliterate the landmarks.

The location of the bevel in relation to the lock on the stylet of the needle is noted, since the use of the bevel in directing the point of the needle has proved to be important. While the lateral borders of the 3rd spinous process are identified by the left hand, the needle is inserted directly in the midline with the bevel placed caudad. The angle toward the patient's head varies with the inclination of the spinous processes, but the needle must never deviate even slightly to either side. The interspinous ligament is then engaged and with the needle held firmly by this structure, the operator's hands are removed and careful inspection is made from the midline to guard against lateral deviation. Further insertion of the needle is not carried out until this alignment is satisfactory. If a tendency to lateral deviation occurs during further progress of the needle, it may be corrected by use of the sheering action of the bevel of the needle, since the point of a needle passing through resistant tissue curves away from the side of the bevel. No pain will be elicited if the needle is in the midline. When the interspinous ligament has been pierced and it becomes evident that the needle is perfectly straight and in the midline, the stylet is removed and the hub of the needle filled with novocain. As the dura is approached, the bevel of the needle is turned laterally in order that the cutting edge of the needle may pierce the dura in the direction of its fibers. It is noted that as the needle point indents the dura, the fluid in the hub of the needle is sucked in by the slight negative pressure created in the epidural space. Careful further insertion pierces the membranes and a free flow of spinal fluid is obtained. The bevel of the needle is then turned caudad and this usually results in a more brisk flow of fluid since the bevel opening now lies between the roots instead of against them. A simple glass-tipped syringe containing one ampule (3.0 cc.) of pantopaque is carefully attached to the hub of the needle. The plunger is withdrawn slightly to aspirate a drop of fluid to insure the presence of a free flow and the pantopaque is then injected with gentle pressure.

Complications of the above procedure are dealt with as follows: *A bloody tap or failure to obtain a free flow of fluid* makes it imperative that the procedure be discontinued because it has been found that continuation under these circumstances almost invariably results in the oil going into the subdural or epidural spaces. Any degree of blood stain in the spinal fluid is considered a bloody tap. When an examination is discontinued for the above reasons, it is postponed for at least ten days, since examination at shorter intervals has resulted in recurrent failure or false defects due to unabsorbed blood or fluid in the subdural or epidural space.

If root pain is obtained, the needle is withdrawn about an inch, the bevel turned to the side of that root and the needle reinserted, using the sheering action of the bevel to direct the needle point toward the midline. If root pain persists, the patient is fluoroscoped and the needle is withdrawn and reinserted to correct any lateral deviation observed.

X-RAY EXAMINATION

The fluoroscopic and radiographic settings that have been found most satisfactory are: 85 kv. with 4 ma. for fluoroscopy, with a change to 50 ma. for spot films. No stationary grid is used because it increases the exposure time, shortens the life of the X-Ray tube and results in no better quality of film than can be obtained without the grid if a small field is used. This field includes the width of the vertebral bodies and varies in length according to the level being examined. On one film both the needle and the top of the sacrum are included for orientation, and all other films include one or the other unless the vertebrae are identified by lead numbers placed under fluoroscopic control. The exposure time varies from 0.3 to ½ second according to the thickness of the patient. A lead letter L is fastened to the back of the fluoroscopic screen at the left margin of the field. The pillow is removed and the fluoroscopic

screen is placed as close to the patient as possible without striking the needle, which is left in place throughout the examination. Some means should be provided to maintain this level if the screen is not provided with a lock. The pool of pantopaque is located fluoroscopically and the table is then tilted head down about 10 degrees. If the pantopaque is in the subarachnoid space, it will flow rapidly and freely, while if subdural the flow is slow and limited. The patient is rolled slightly to one side or the other, if necessary, to align the spinous processes to the center of the vertebral bodies. This requires complete accommodation of the radiologist's eyes, particularly with heavy patients. The patient is not allowed to cough when the table is tilted foot down because this seems frequently to render removal difficult, due, theoretically, to escape of oil or fluid around the needle under the sudden increase of pressure. Examination is started at the 3rd interspace unless a higher lesion is indicated clinically, and the 4th and 5th discs are then examined. The root sheaths seem to fill more completely when the pantopaque is running caudally instead of cephalad. Two views are taken of each disc, one with it partially covered,[2] and the other with it completely covered by the pantopaque. The central extension of a disc may be obscured by the center of the pantopaque column but outlined by the thinner end of the column. Oblique views are taken of the suspicious levels and give added courage in making a diagnosis but have in no instance disclosed a disc not demonstrated by the above views. Lateral views were taken in about 100 cases but added no additional information and were discontinued.

On completion of the roentgenological examination, the table is tilted until the needle point is in the center of the pantopaque column for removal. If the pantopaque obscures the needle, the patient may be rolled slightly to one side to ascertain the level of the point. It the point is not in the midline, the patient is rolled 10 to 20 degrees toward the side of the point before removal is attempted.

REMOVAL

The pantopaque syringe is again carefully attached to the needle and withdrawal is accomplished by gentle suction. This may be best described as enough pull on the plunger of the syringe to overcome the effects of gravity. This gentle pull is continued until fluid streams up through the pantopaque without further increase in the volume of pantopaque in the syringe. The patient is again fluoroscoped and any remaining oil centered to the needle point. It may be necessary to again roll the patient to one side or the other to accomplish perfect centering. The aspiration procedure is repeated, until no further oil can be seen fluoroscopically. The needle is then withdrawn and the needle site slightly massaged. If there is any bleeding at the needle site, a square of gauze is applied with adhesive. The patient then gets off the table, puts on his clothes and walks back to his ward. It has been our experience, as that of others,[7] that this regime has resulted in fewer post-lumbar-puncture headaches than when the patient is treated as a litter case. The rare headache that develops is treated symptomatically by bed rest.

Complications of withdrawal are dealt with as follows: *If the patient has root pain*, the bevel of the needle is rotated either 180 or 90 degrees away from the side of the pain. *If no flow is obtained*, the needle is rotated very freely for several turns and suction is applied with the bevel at a different angle. *If the pantopaque runs past the needle or is divided* at the level of a prominent disc, sometimes it may be collected around the needle point by placing a pillow under the chest or upper thighs to increase the concavity of the lumbar curve. *If clear fluid is obtained* when the needle point is in the midline and centered to the pantopaque column, it must be assumed that it lies in the spinal fluid supernatant to the pantopaque pool. Under this circumstance, the pantopaque may be reached by very slight further insertion of the needle. *If there is a filling defect* around the needle point, presumably due to blood or fluid in the adjacent subdural or epidural space or due to a disc, there may also be a free flow of fluid without pantopaque. Under this circumstance, the patient is rolled 45 degrees toward the side of the needle point and pantopaque is occasionally obtained. *If the above maneuvers are unsuccessful*, time is saved by inserting another needle at a lower interspace that is free of a filling defect. The first needle, under these circumstances, is never taken out until removal

is completed, because if the needle is withdrawn, oil or fluid frequently escapes through the needle hole into the subdural or epidural space and renders success of the second tap less likely.

CERVICAL MYELOGRAPHY

In cervical myelography several modifications of the above technique are employed. Two ampules (about 6.0 cc.) of pantopaque are used. With this amount of contrast medium a diagnostic quantity can be made to flow into the cervical region with less danger of its entering the basal cisterns. Also the larger quantity gives better delineation of the nerve roots. Injection is made at L-4 or L-5 for greater ease in removal. The pillow is removed from under the patient, the table is tilted to the horizontal position, and the patient is then pivoted on his abdomen until his head is at the foot of the table.

For non-paralyzed patients the following procedure is employed. The foot rest is moved up near the end of travel of the fluoroscopic tube and a small bolster (or sand bag) placed on it as a cushion on which the patient can rest his elbows to support his weight when he is tilted head downward. He is moved up or down the table until he can rest his elbows against the cushion and at the same time rest his chin on his doubled fists, placed one on top of the other to force his head as far back as possible without raising his chest from the table. The degree of hyperextension varies with different patients and controls the angle to which the table can be tipped safely.

After the patient is firmly braced in this position, the table is tilted slightly beyond the point at which the lumbar concavity lies above the dorsal convexity. To increase the speed of flow, the tilt may be increased further but not to the point where the patient's ears are below the level of his shoulders. The flow of pantopaque is observed fluoroscopically past the dorsal curve until the major portion has passed, usually a period of 10–15 seconds. No attempt is made to pass the few drops that usually trail the main mass. Early attempts to prevent breaking up of the pantopaque column by a gradual tilt were discontinued when it was found that the column broke up in any event.

As soon as the major portion of pantopaque has entered the cervical region, the table is tilted back toward the horizontal until the pantopaque reaches the first thoracic vertebra. The patient's head is then lowered manually but kept hyperextended until his chin touches the table top. The patient is never allowed to move his own head, because he is apt to lower it too far and allow pantopaque to flow into the basal cisterns.

Spot films are taken under fluoroscopic control as in lumbar myelography except that the exposure time is shortened to 1/10–2/10 second and larger fields are used to include the first ribs on all films for localization. The spread of pantopaque in the cervical region is such that films taken at two different degrees of tilt will usually delineate the entire region. Oblique views were taken in earlier examinations but have been discontinued since they seemed to offer no additional information over true A. P. views. More clear films can sometimes be obtained by using an overhead tube with fast Bucky technique.

To remove the pantopaque the patient is pivoted back to the position of injection, with the table horizontal and his head held in hyperextension by an assistant. The table is then tilted until the patient is standing. The basal cisterns, cervical and dorsal areas are then inspected fluoroscopically for droplets of pantopaque. Any such droplets can usually be brought down by gently shaking the patient. The patient is told not to cough because this has seemed to make removal more difficult, as previously stated. The pantopaque is globulated after cervical myelography and removal is easier if the needle is at L-5 and the globulated mass can be collected in the caudal sac.

For patients who are paralyzed or badly disabled the same general principles are employed as with active patients, the only difference being that immobilization and manipulation are done by assistants instead of the patient. As in other phases of myelography, the procedure is kept as simple as possible and the use of elaborate or complicated apparatus avoided. The standard immobilizing band which comes with the table is used to prevent sliding of the patient when his head is tilted down. This band is tightened over the patient's lower thighs,

anchored by wooden pegs or wedges and the patient suspended as from a trapeze, with one assistant holding his legs doubled over the band and another holding the patient's head.

RESULTS

Difficulties were encountered in approximately 10 per cent of the examinations performed by several different officers in this series of over 675 myelograms. These were usually due to bloody taps or subdural injections. Although these may sometimes occur with the most careful technique, the incidence in this series became markedly reduced as the importance of the various details, described above, became appreciated. In each incidence of failure, a successful examination has been possible at a later date.

The average time required to perform a myelogram should be about 20 minutes. On two occasions, in which the time interval was noted in this series, 13 examinations were performed in 5 hours and 6 examinations in 2 hours, respectively. The shortest measured time consumed to complete one examination was 8 minutes, while a careful cervical myelogram usually requires more than 30 minutes.

Removal is practically complete in nearly all instances. A few minute droplets frequently remain because they cannot be seen fluoroscopically and therefore cannot be centered to the needle point. If removal is difficult, the last $\frac{1}{2}$ cc. is allowed to remain rather than to subject the patient to any unnecessary discomfort, since small amounts of pantopaque are absorbed.[12] In no instance during the past year has it been found necessary to leave a greater quantity than $\frac{1}{2}$ cc. in the subarachnoid space. In some instances, 50 to 90 per cent of the oil injected subdurally has been removed without moving the needle through which it was injected. The approach to and the handling of the examination as routine rather than a formidable procedure has resulted in a marked decrease of the apprehension with which the patients regard the examination and has been associated with almost complete disappearance of post-lumbar-puncture headaches.

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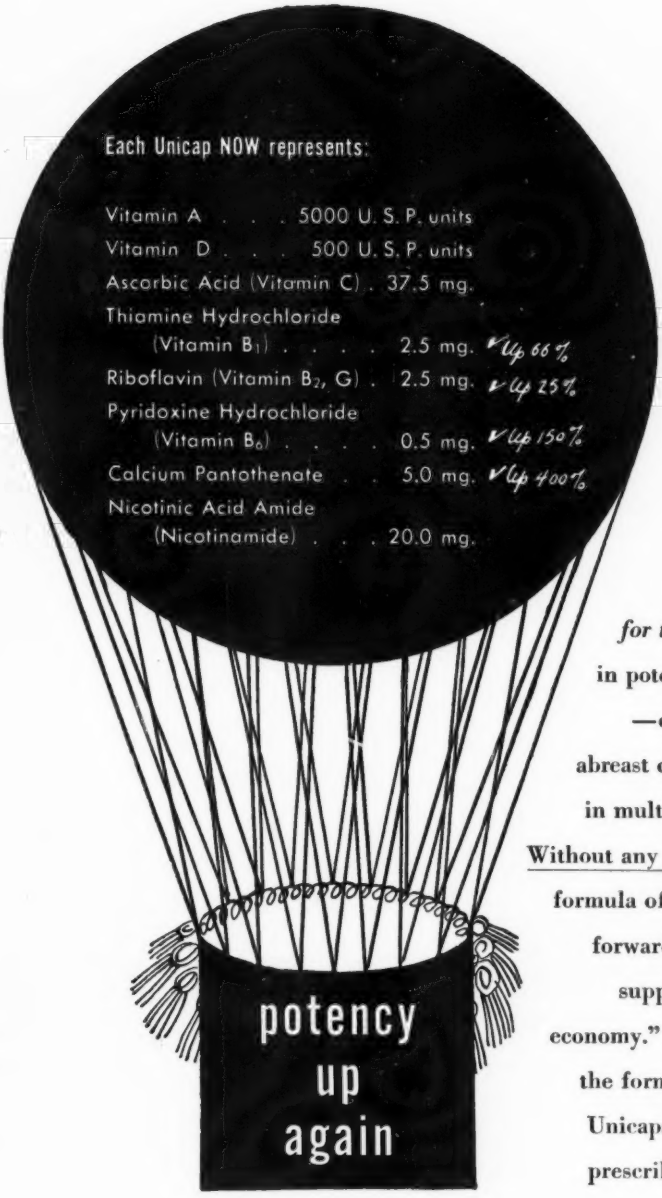
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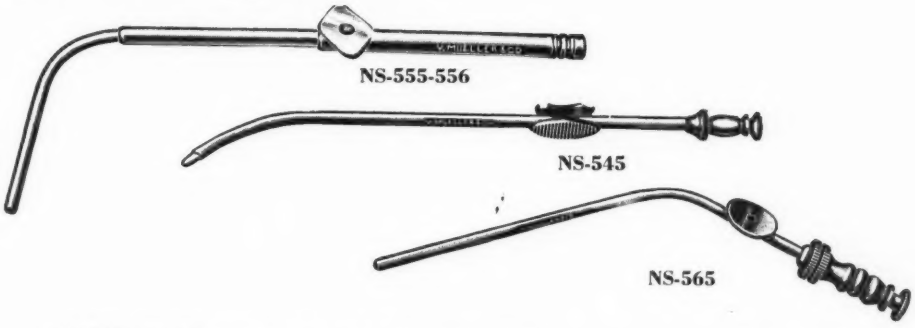

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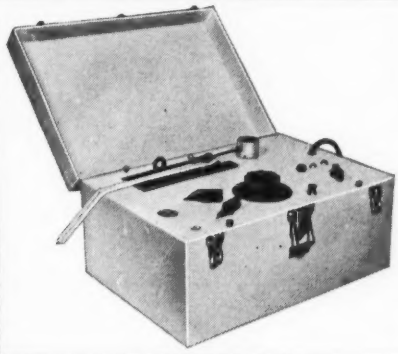

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HISTOLOGIC STUDIES OF THE BRAIN FOLLOWING HEAD TRAUMA

II. POST-TRAUMATIC PETECHIAL AND MASSIVE INTRACEREBRAL HEMORRHAGE

JOSEPH P. EVANS, M.D., AND I. MARK SCHEINKER, M.D.

The Laboratory of Neuropathology, Cincinnati General Hospital, and the Department of Surgery, University of Cincinnati College of Medicine, Cincinnati, Ohio

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THE CUT BRAIN as seen at the autopsy table following severe head trauma raises a two-fold question: "What is the mechanism of formation of the various types of hemorrhage seen?", and secondly, "What are the anatomic and physiologic end results of similar, but less severe changes in the brain of the patient who survives injury?"

There are several different types of hemorrhage resulting from trauma to the head. The first is that due to cortical laceration, occurring, according to Martland and Beling,[19] in about 82 per cent of cases. A second type, deep massive hemorrhage, was designated by Kolisko[16] as the large intramedullary type (Markblutung). A third consists of multiple minute punctate hemorrhages distributed chiefly in the white matter, especially in the centrum semi-ovale. This last type assumes two forms: (a) petechial hemorrhages filling the Virchow-Robin spaces, and (b) "ring hemorrhages" with hemorrhagic infiltration of the perivascular adjacent tissue. Finally, there is a fourth type consisting of perivenous extravasations in the midbrain.[31]

Our knowledge of the precipitating mechanism of these hemorrhages, with the exception of the first, which is satisfactorily explained by tearing of the blood vessels,[19] and of the fourth,[31] is very limited. It is the purpose of this paper to help clarify the pathologic physiology underlying the changes seen in the other forms of traumatic hemorrhage, and hence, to seek an answer to the first question proposed in the introduction. The answers to the second question have been sought in another study.[8]

Jakob[15] demonstrated experimentally that hammer blows over the parietal bone resulted in mild parietal lesions and much more severe remote changes in the midbrain, brain stem, and even the upper cervical cord. Scagliosi[26] has made similar observations, as had also Pfeifer[21] in his study of experimental puncture wounds. These observations serve to emphasize the importance of changes in areas remote from the site of initial injury.

Massive hemorrhage deep in the cerebral hemispheres has been described by Kolisko,[16] Schwarzacher,[33] and by Reuter.[24] Similar observations have been made by Marburg,[17,18] Friedman,[14] Fey,[12] Renoux,[23] Stenvers,[34] Wilson and Winkelman,[36] Caló,[3] Crouzon,[5] and by many others.

Petechial hemorrhages have been studied by Schmaus,[32] Jakob,[15] Ricker,[25] Berner,[2] Cassasa,[4] Osnato and Giliberti,[20] Martland and Beling,[19] Winkel-

man and Eckel,[37] Symonds,[35] Rand and Courville,[22] Schaller, Tamaki and Newman,[27,28] and many others. Related experimental studies have been made by Allen,[1] and Ferraro.[10]

MATERIAL

The conclusions drawn in this paper are based on a study of eight cases presenting varying grades of post-traumatic hemorrhage, not directly related to the area of coup or contrecoup injury. Only three of these cases will be presented in detail, though brief notes of the others will be given.

PRESENTATION OF CASES

Case 1. *A 53-year-old male. Blow in the occipital region. Death approximately 24 hours later. Necropsy. Cortical and subcortical hemorrhage. Periventricular ball hemorrhages and multiple midbrain hemorrhages.*

History. J. A., a 53-year-old white male, was admitted to the hospital about 18 hours after receiving a blow on the back of his head.

Examination and Course. On admission he was unconscious, showed signs of decerebrate

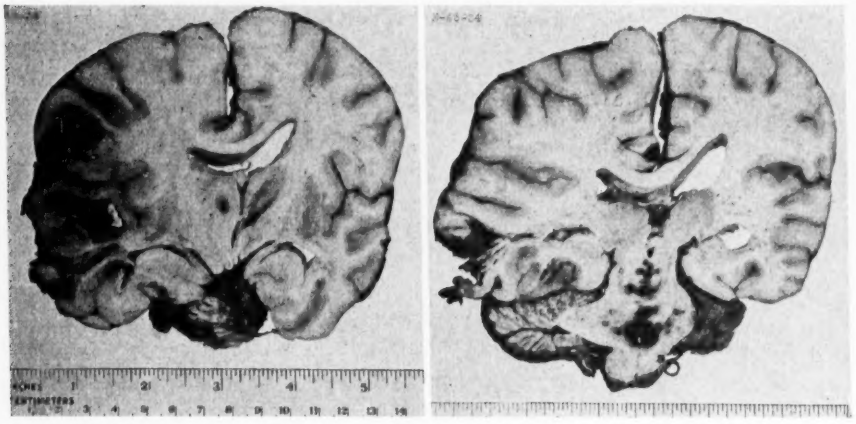

FIG. 1 (*left*). Case 1. Hemorrhages in the distribution of the right middle cerebral artery, ball hemorrhages in the optic thalami, and hemorrhagic softenings in the right cingulate and right hippocampal regions.

FIG. 2 (*right*). Case 1. Multiple midbrain hemorrhages.

rigidity, and a fixed dilated pupil on the right. The decerebration had been present for some 8 hours previously. Blood pressure was 135/80, the pulse 80, respirations 18.

A right subtemporal burr opening was placed without delay, revealing a small extradural and a massive subdural accumulation of blood. Bilateral frontal and right occipital openings were also made. There was no blood found over the left hemisphere. During the procedure the patient lightened somewhat for a brief period, but thereafter his postoperative course was steadily downhill, all efforts designed to relieve the intracranial pressure failing. The respirations ceased about 24 hours after the injury, though with the maintenance of artificial respiration the heart was sustained for another 4 hours.

Necropsy was performed 5 hours postmortem. There were fractures of the greater wing of the right sphenoid bone and of the parietal and temporal bones. Small residual accumulations

of extradural clot were found in the right frontal and occipital regions, and there was recurrent subdural clot over the right frontal lobe.

Gross Anatomic Changes. The entire right hemisphere was swollen and there was a zone of hemorrhagic softening confined to the distribution of the right middle cerebral artery with an area of tissue destruction involving the middle portion of the second and third temporal convolutions. The inferior portions of both frontal lobes were markedly contused. There was a herniation of the uncal portion of the right temporal lobe, measuring 1.5 cm. in length and 15 mm. in width. The midline structures, particularly the optic chiasm, were slightly shifted to the left.

Coronal section through the hemispheres (Fig. 1) showed a swelling of the right with compression of the lateral ventricle of this side and shift of the third ventricle to the left.

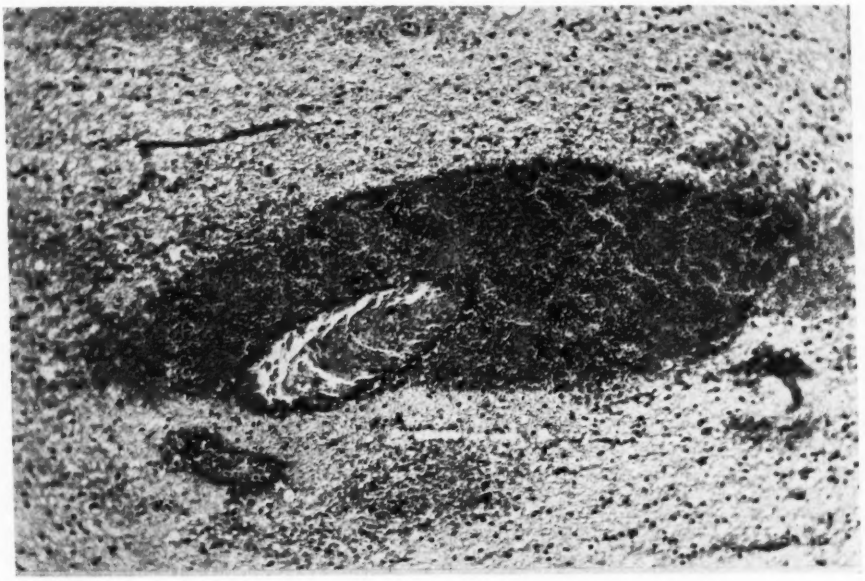

FIG. 3. Case 1. Small blood vessel, probably a vein, showing evidence of vasoparalysis. Note distention of perivascular space by extravasated blood. Hematoxylin and eosin, ×125.

There were numerous areas of massive and petechial hemorrhages involving the subcortical white matter of the third frontal and the first and second temporal convolutions of the right hemisphere. The cortical ribbon was fairly well preserved. There were ball hemorrhages present in the optic thalamus on both sides. The right hypothalamic region appeared swollen and was displaced downward through the tentorial opening. In addition there were small areas of hemorrhagic softening in the herniated portion of the right cingulate gyrus and the herniated right hippocampal region.

Fig. 2 shows clearly the numerous small hemorrhages observed in the peri-aqueductal region and the vicinity of the pontine raphé.

Microscopic Observations. Sections from the central white matter and the basal ganglia disclosed lesions characterized by tremendous distention and stasis of the smaller veins and capillaries associated with degenerative changes and increased permeability of the vessel wall for red blood cells. These are changes which we have come to regard as typical of vaso-

paralysis.[29] The majority of the smaller hemorrhages were found to contain one or several blood vessels surrounded by large masses of transudated blood (Fig. 3). These vessels, chiefly veins of medium size, were congested, and their walls disclosed varying degrees of degenerative alteration, even to necrosis, when the almost complete disintegration of the vessel wall resulted in the merging of intravascular blood with the extravasated blood enveloping it (Fig. 4). In some instances it was difficult to be sure of the nature of the vessel involved, so complete was the necrosis of the vessel wall (Fig. 5).

Sections taken from the brain stem showed comparable changes of varying degree, often disclosing clearly the relationship to tremendously distended veins. In the vicinity of almost all of the hemorrhages engorged veins were surrounded by large masses of extravasated erythrocytes distending the perivascular spaces.

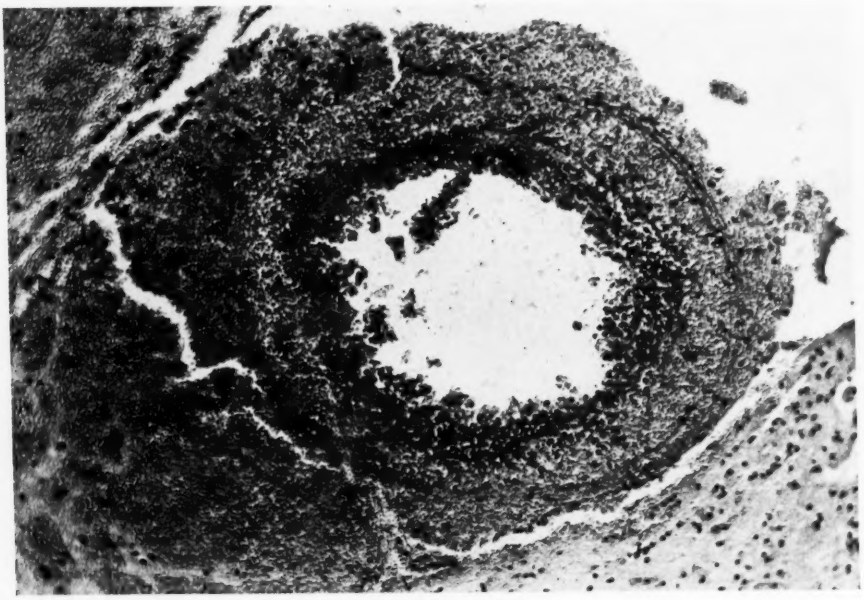

FIG. 4. Case 1. Necrotic vein. Hematoxylin and eosin, ×170.

The nervous parenchyma revealed rarefaction and edema formation, particularly in the immediate vicinity of the hemorrhages (Fig. 5).

Loyez stains of the myelin sheaths showed numerous focal areas of myelin loss in the vicinity of the congested veins.

In addition there were numerous small foci of tissue necrosis, just as are seen in anemic infarction. There was no glial reaction, nor was there proliferation of phagocytes.

Case 2. *A 66-year-old male. Fall from second-story window. Severe head and chest injuries. Coma. Death in three days. Necropsy. Massive and petechial intracerebral hemorrhage.*

History. J. M., a 66-year-old colored male, was admitted to the hospital about ½ hour after he had fallen from a second-story window. He was immediately rendered unconscious and never regained consciousness.

Examination and Course. Initial examination in the Receiving Ward showed a hematoma

of the scalp in the right occipitoparietal region, generalized flaccidity and complete areflexia, a crushed right chest, blood pressure 125/65, and pulse 80.

Following admission to the ward, there was found a left hemiparesis involving the face as well as the extremities. Lumbar puncture revealed a grossly bloody fluid (275,000 red blood cells per c. mm.), under an initial pressure of 110 mm. of water. The next day, the initial pressure was 120, and there were 390,000 red blood cells. Skull roentgenograms showed a small depression in the right occipital region with extension of a linear fracture into the base.

The diagnoses were made of crushed chest, fracture of the skull, cerebral concussion and

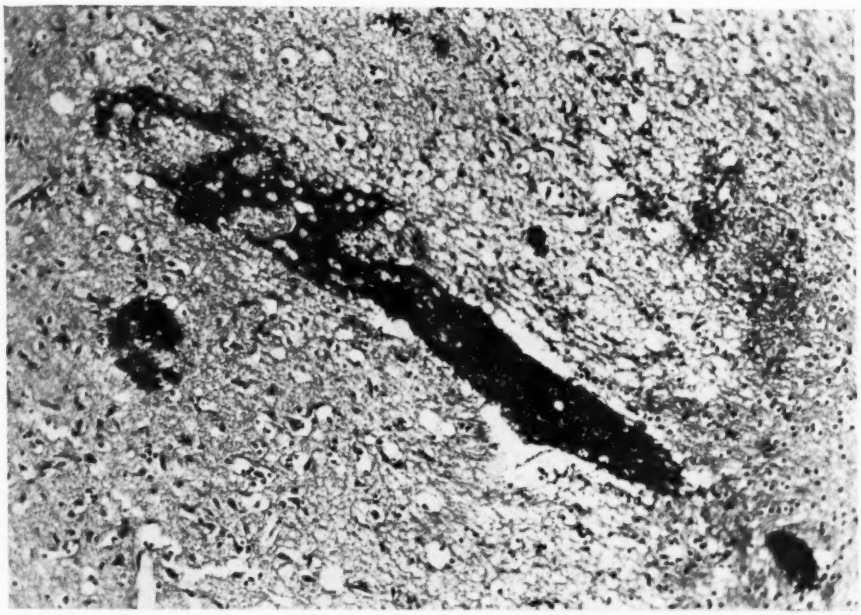

FIG. 5. Case 1. Necrotic vessel surrounded by edematous tissue. Hematoxylin and eosin, ×125.

contusion, and subarachnoid hemorrhage. The patient survived 3 days. At no time was operation deemed advisable.

Necropsy. This was performed 4 hours postmortem. There were fractures of the left 6th and 7th ribs and of the sternum with intrapleural and subpleural laceration on the left. The left pleural cavity contained 1400 cc. of blood, the right 1000 cc. There was left pulmonary atelectasis. There were found a miminal depressed skull fracture in the right posterior parietal region, and a linear fracture extending into the right temporal region. An extradural clot, only 1 to 2 mm. in thickness, was over the right occipitoparietal area. A minimal subdural collection of blood was over each parietal lobe.

Gross Anatomic Changes. The brain showed diffuse subarachnoid hemorrhage and no evidence of increased intracranial pressure. There was extensive tearing of cerebral tissue at the right occipito-parieto-temporal junction. In addition, there was a second large defect on the lateral surface of the anterior two-thirds of the left temporal lobe.

The cerebrum was sectioned coronally (Fig. 6). The cerebellum and brain stem were cut horizontally. All of the cerebral sections were somewhat soft, displaying a slight degree of

edema. There were numerous scattered petechial hemorrhages throughout the white matter, particularly in the region of the centrum semi-ovale. The right hemisphere was fuller than the left. Lying in the right optic thalamus and compressing the body of the right lateral ventricle was a blood clot measuring 2 cm. in length and 1 cm. in diameter. About the periphery of the gross hemorrhage was a zone of punctate bleeding, ranging from 5 to 10 mm. in thickness. The midline structures were displaced to the left. Eight sections of the brain stem showed a moderate degree of congestion and petechial hemorrhage, especially about the aqueduct of Sylvius and the fourth ventricle.

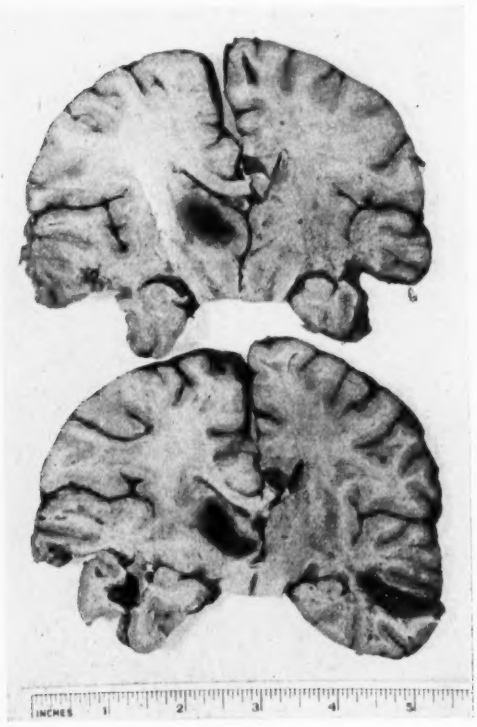

FIG. 6. Case 2. Massive hemorrhage in the right optic thalamus; petechial hemorrhages diffusely scattered throughout white and gray matter.

Microscopic Observations. The following alterations relevant to the present problem were observed:

(1) Massive hemorrhage. The histologic details of this phenomenon need not be further elaborated since they are common to massive hemorrhage occurring under other circumstances.

Rather are we concerned with the forms of petechial hemorrhage that we have observed.

(2) Ring hemorrhages. Many of the petechial hemorrhages were found under low magnification to be characterized by the changes shown in Fig. 7. These hemorrhagic areas had a clear, pale-staining center, surrounded by a dense ring of extravasated blood. The centrum of the clear-staining area contained a slight accumulation of amorphous detritus, some of it being blood pigment. Sometimes the centrum revealed a small blood vessel with degeneration or necrosis of the vessel wall.

(3) Perivascular hemorrhages. Other petechial hemorrhages showed congested venules and capillaries surrounded by a large conglomeration of erythrocytes, generally limited to the distended fibrin-containing perivascular spaces. In some instances, the distention was so great that rupture of the limiting membrane had occurred with extravasation of the red blood cells into the contiguous brain parenchyma. There was no inflammation. The vessel walls themselves exhibited degeneration and necrosis.

(4) Morphologic equivalents of functional vascular disturbances: In addition to the changes noted in (2) and (3), many of the vessels exhibited the signs of vasoparalysis associated with considerable distention of the perivascular spaces with serous fluid. In some instances the adjacent cerebral tissue disclosed liquefaction and edema. In such areas, distributed throughout most of the white matter, the majority of the capillaries were distended with blood and showed signs of congestion and stasis.

(5) Necrosis of the vessel wall: In sections taken from the basal ganglia immediately adjacent to the massive hemorrhage, the vascular alterations were even more marked than those described immediately above. Here some small vessels revealed the characteristic find-

ings of vasothrombosis[30] and rare ones were actually occluded by fibrin thrombi. The vessel walls revealed degenerative changes and the surrounding perivascular tissues showed signs of disintegration, an obvious indication of impaired circulation with resultant hypoxia (Fig. 8).

Case 3. *A 33-year-old male. Head injury of undetermined mechanism. Left skull fracture and left-sided paresis. Exploration and partial evacuation of right-sided subdural hematoma. Death 6 days after injury and 2 days after exploration. Necropsy. Contrecoup damage of right temporal and frontal lobes, gross hemorrhage in the splenium, and diffuse petechial hemorrhages in the white matter of both hemispheres.*

History. G. deP., a 33-year-old colored male, was admitted to the hospital on June 18, 1942. No story of his accident was available.

Examination and Course. Examination on admission showed signs of acute alcohol poison-

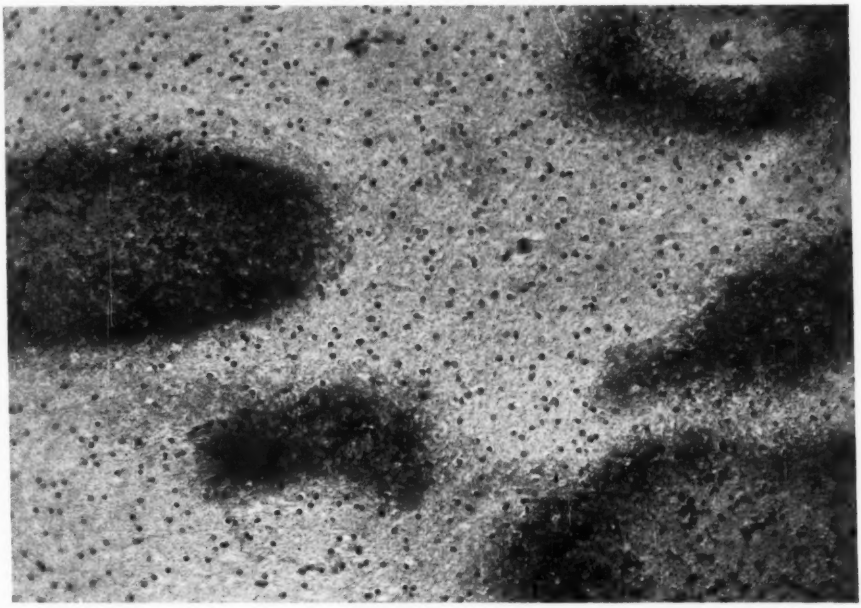

FIG. 7. Case 2. Ring hemorrhages with a pale-staining center surrounded by a dense ring of extravasated blood. Hematoxylin and eosin, ×110.

ing. He could not be aroused and his pupils were 3 mm. in diameter, and reacted to light. There was a left-sided paresis and the deep reflexes were depressed on the left. The superficial reflexes were absent. There was bruising of the head in the left posterior parietal region. His blood pressure was 160/110, pulse 80, respirations 20.

Laboratory studies on admission revealed sugar in the urine, a blood sugar level of 187 mg. per 100 cc., carbon dioxide combining power of 49 vol. per cent, and blood urea nitrogen of 17 mg. per 100 cc. His blood Wassermann reaction was negative. X-rays of the skull showed two left occipitoparietal linear fractures.

On the day following admission, his conscious level had improved somewhat, but he was restless and uncooperative. A defect was suspected in the left homonymous visual fields. The

left-sided weakness persisted. Lumbar puncture showed an initial pressure of 150 mm. of water and a grossly bloody fluid.

The succeeding day frequent convulsive movements developed in the face and in the arms which were more marked on the left. He became duller and his temperature ranged around 103°. Multiple exploratory burr holes revealed a subdural hematoma on the right and extensive right temporal lobe contusion. Thereafter his course was progressively downhill and death occurred 6 days after admission and 2 days after operation.

Necropsy. This was performed 8 hours postmortem. No significant factors contributing to the death were found in the autopsy except as noted below.

Gross Anatomic Changes. The brain showed an epidural hematoma and an extensive subdural hematoma over the lateral surface of the right frontal region. The latter measured

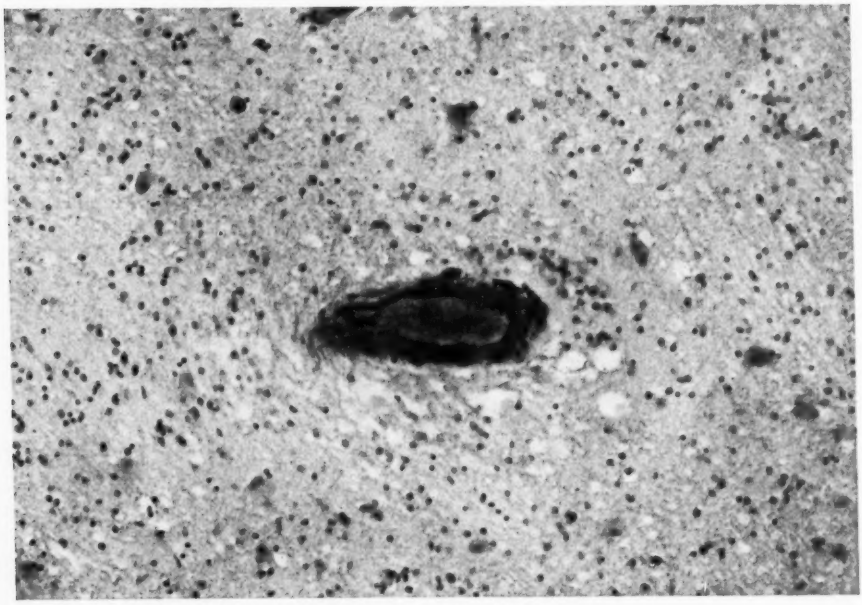

FIG. 8. Case 2. Degeneration of blood-vessel wall and early perivascular edema. Hematoxylin and eosin, ×160.

8 mm. in thickness and extended posteriorly across the Sylvian fissure, covering the anterior portion of the first and second convolutions. Subarachnoid hemorrhage was present over both parietal areas. The right hemisphere was fuller than the left, the gyri were broadened, and the sulci narrowed. There was a well defined uncal herniation of the right temporal lobe.

Multiple coronal sections of the hemispheres showed contusion of the right lower parietal and upper temporal regions, small petechial hemorrhages scattered throughout the white matter of both hemispheres, and two areas of massive hemorrhage, one in the right temporal lobe and the second in the right side of the splenium of the corpus callosum. Sections through the midbrain and cerebellum showed congestion of the vessels of the white matter.

Microscopic Observations. Blocks for histologic study were taken from the right temporal and frontal lobes, the corpus callosum, the midbrain, and the cerebellum. They were stained by the hematoxylin and eosin, hematoxylin and Van Gieson, and cresyl violet techniques, and were impregnated by the silver method of Bodian.

Temporal lobe: These sections showed engorgement of the subarachnoid spaces by hemorrhage. The leptomeningeal vessels were distended with blood and some of the pial vessels showed white thrombi. In the gray matter was massive hemorrhage, surrounded at the periphery by scattered petechial perivascular and ring hemorrhages extending into the white matter. The vessels of these areas were engorged and dilated, and their walls frequently showed degeneration and necrosis. The parenchyma was severely damaged, many nerve cells being degenerated or destroyed. Many of the astrocytes showed swelling and ameboid transformation. The oligodendroglia appeared to be relatively little affected, and in the sections impregnated by the Bodian silver method many nerve fibers appeared slightly degenerated, showed moderate fusiform swelling, and were separated by serous exudate.

Frontal lobe: These sections showed massive subarachnoid bleeding. There were found in the cortical and subcortical areas congestion of the vessels and distention of the pericellular and pericapillary spaces. The glial cells in the white matter showed varying grades of astrocytic change from slight swelling to ameboid degeneration. Within the cortex there was slight proliferation of microglia.

Corpus callosum: There were numerous changes present in these sections. Diffusely scattered petechial hemorrhages of the ring type were seen about congested blood vessels. A large area of massive hemorrhage was formed by the apparent coalescence of such hemorrhages. The basis for the petechial hemorrhages was to be found presumably in the stasis observed in the capillaries and veins. In many instances, not only the vessels, but also the perivascular spaces were distended. They usually contained serous fluid or extravasated red and white blood cells. In addition there were degeneration and necrosis of the vessel walls. In some instances congested vessels were surrounded by small areas of early edema formation. The parenchyma appeared rarefied, with signs of liquefaction and degeneration, and occasional foci of small infarctions were seen both in association with and in the absence of hemorrhages. About the smaller hemorrhagic areas there were regressive changes of the macroglia. Only occasional gitter cells were present.

Case 4. *Summary.* L. G., a white man of 55, was injured when his car was sideswiped by another car. Examination showed a hematoma of the scalp in the right temporal region, scalp lacerations over the vertex and the left eyebrow, coma and no localizing signs necessitating operation. X-rays of the skull showed a linear fracture in the right temporal region, running to the vertex. Lumbar puncture revealed a pressure of 250 mm. of water, and a moderately bloody fluid. Diagnoses of cerebral concussion, cerebral contusion, and subarachnoid hemorrhage were made. He lived for a week following the injury.

Necropsy was limited to the head. The right hemisphere was a little fuller than the left. The right ventricle was somewhat compressed. The midline was shifted slightly to the left. Punctate hemorrhages surrounded the area of extensive right temporal lobe destruction.

Survey sections for microscopic examination were taken from the basal ganglia and the cornu Ammonis. They showed ring and petechial hemorrhages, especially in the putamen. The small veins and capillaries appeared congested and engorged with blood. Their walls revealed moderate signs of degeneration. The nervous tissue showed rarefaction and edema.

Case 5. *Summary.* E. S., a man of 30, fell from a third-story roof and sustained multiple bodily injuries, including a depressed fracture of the right fronto-parieto-temporal region, and several fractures of the extremities. He died about 2 hours after the injury.

Necropsy confirmed the presence of an extensive skull fracture, multiple fractures of the extremities, and perinephritic and suprarenal hemorrhages. There were massive subdural hemorrhages and extensive subarachnoid bleeding.

Coronal sections showed, in addition to the superficial tearing of cerebral substance, petechial hemorrhages scattered throughout the gray and white matter, chiefly in the inferior half of the brain.

Survey sections for histologic study taken from the basal ganglia, the cornu Ammonis, and the hypothalamus showed congestion of the veins and capillaries, vessel wall degeneration, maximal distention and some disruption by red blood cells of the perivascular spaces, coales-

cence in some areas of perivascular extravasations, and edema of the nervous tissue adjacent to the blood vessels. No arteriosclerotic changes were observed.

Case 6. *Summary.* T. M., a 64-year-old man, fell down a flight of steps and was rendered unconscious. X-rays of the skull revealed a linear fracture in the right temporoparietal region. There were no localizing signs, but a subdural hematoma was suspected and sought on the right side. It was only at autopsy, 36 hours after the injury, that a large left-sided subdural hematoma was found.

Examination of the cut brain showed swelling of the left hemispheres, involving the corona radiata and the basal ganglia, and causing compression of the left lateral ventricle and shift of the midline structures to the right. In addition there were scattered coalescent hemorrhages and diffuse petechial hemorrhages within the basal ganglia.

Survey sections for histologic study were taken from the centrum ovale of the right hemisphere and from the area adjacent to the right lateral ventricle. They showed small perivascular hemorrhages. Some of the blood vessels showed a moderate degree of degeneration of their wall and these were surrounded by large masses of red blood cells. The nervous tissue exhibited distention of the pericellular spaces.

Case 7. *Summary.* B. R., a woman of 59, was admitted in a comatose condition without obtainable history. There were no localizing neurologic signs. The deep reflexes were present in the upper extremities and absent in the lower. The abdominal reflexes were absent; the Hoffmann and Babinski reflexes were bilaterally positive. Lumbar puncture revealed a bloody fluid under an initial pressure of 350 mm. of water. Surgical exploration seemed not indicated. Death occurred 12 hours after admission to the hospital.

Necropsy showed a hematoma of the scalp in the left temporal region, a linear skull fracture in the left parietal bone, and an extensive right subdural hematoma. There were gross destruction of the right temporal lobe, massive right peri-Sylvian and cingulate gyrus hemorrhages; and swelling of the right hemisphere with obliteration of the right lateral ventricle and shift of the midline structures to the left. Petechial hemorrhages were scattered diffusely throughout both the gray and the white matter, especially in the white matter of the frontal lobes, and the gray substance of the basal ganglia.

Survey sections for microscopic study were taken from the basal ganglia, corpus callosum, and cerebellum. These revealed the presence of diffusely scattered petechial hemorrhages associated with distention and congestion of the blood vessels. In some areas there was an accumulation of serous fluid and red blood cells within the distended perivascular spaces. There was edema of the nervous tissue.

Case 8. *Summary.* D. B., a man of 42, fell while intoxicated and suffered a severe head injury. Admission examination showed no localizing neurologic signs. A grossly bloody spinal fluid was found under an initial pressure of 250 mm. of water. X-rays of the skull showed a linear fracture of the cranial vault on the left. About 24 hours after injury there developed deepening coma. Localizing signs were equivocal and operation was postponed until approximately 48 hours after injury, when a right subtemporal decompression revealed a small extradural collection of blood and the underlying cortex was found to be badly lacerated. He improved temporarily but on the 3rd day after injury he again became comatose, and a small left subtemporal opening was made with the finding of only a small amount of subdural clot. The next day his lumbar puncture pressure was 240 mm. of water. He gradually lost ground and died 7 days following his injury. It should be noted that his blood Wassermann was negative.

Necropsy revealed an epidural hemorrhage (2 cm. in thickness and 9 cm. in diameter) in the right occipital region and a bilateral ragged laceration of the surface of the temporal lobes. In addition, there were small areas of massive hemorrhage in the vicinity of the right lateral ventricle and petechial hemorrhages diffusely scattered throughout the white substance of both hemispheres. There was a well defined uncal herniation on the right.

Survey sections for microscopic study were taken from the basal ganglia and from the white matter superior and lateral to the caudate nucleus on the left side. There were found numerous diffusely scattered perivascular ring hemorrhages. In addition, there were many circumscribed small petechial hemorrhages, and advanced rarefaction and edema formation of the brain parenchyma.

COMMENT

We have presented examples of petechial and massive hemorrhage occurring as the result of head injury. The local and superficial changes occurring at the site of the blow and at the site opposite the injury (coup and contrecoup) have an obvious mechanical explanation. We wish, however, to draw attention to certain features in our cases of intermediately placed hemorrhages, petechial and massive, which have the following points in common:

(1) Scattered lesions, characterized by liquefaction and edema formation in the parenchyma, changes believed due to hypoxia.

(2) Less extensive changes represented by perivascular hemorrhage and perivascular edema. That the latter change does not represent fixation artifact is borne out by the third type of change.

(3) Significant alterations of the vessels themselves, characteristic of vasoparalysis[29] and vasothrombosis.[30]

(4) Massive hemorrhages which we believe result, in some instances at least, from the coalescence of multiple areas of diapedetic hemorrhage.

It is quite possible that the changes noted in Case 1 in the distribution of the middle cerebral artery may represent hemorrhagic infarction of that vessel, a mechanism described in another paper in this series.[7] But the presence of alterations deep within the cerebrum seems of itself to be sufficient indication of the transmission of force throughout the cerebral substance. While it is conceivable that in some instances vascular changes may result from tearing of the deeper vessels, we believe that there occur in addition profound disturbances that result from mechanically produced vasoparalysis. The work of Florey,[13] and recently that of Echlin,[6] on the vascular responses to trauma inflicted on the surface vessels of the brain can probably be translated to the deeper vessels, inaccessible to present methods of experimental study.

These disturbances, we postulate, result in stasis and vasoparalysis, and these in turn lead to perivascular edema, and later to perivascular hemorrhages, which may give rise to petechial hemorrhage and in some instances to coalescent small hemorrhages which form massive accumulations of blood. Concurrent nutritional changes due to hypoxia further increase the permeability of the vessel wall. Undoubtedly, expanding zones of hemorrhage cause local regressive changes in the glial tissue (clasmatodendrosis and acute swelling of the oligodendroglia) and the process may pass from a reversible into an irreversible one, causing irreparable structural changes.

It must be recognized that direct proof for the existence of propagated vasomotor disturbances in the depths of the cerebral tissue is lacking, though Ricker[25] had adduced strong evidence in favor of it. Except for the mechani-

cal rupture of deep vessels, which we believe to be only occasional, we see no other satisfactory explanation for the changes occurring deep in the cerebral substance, and we believe that such an hypothesis explains satisfactorily the changes that we have described above. It should be borne in mind that in association with severe forms of head injuries, there are profound alterations in the brain stem mechanisms controlling blood pressure, pulse, respiration, and vasomotor tone in the body generally. Such alterations, we have reason to believe from unpublished work of Ferris[11] and his co-workers, may at times, in the more severe injuries, produce a significant impairment in cerebral circulation. Under such circumstances, the local changes resulting from the postulated mechanism may well be heightened by general impairment of intracranial circulation. Likewise injury to the pulmonary or circulatory systems proper may serve to accentuate alterations due to direct brain trauma.

SUMMARY

1. Three cases of post-traumatic petechial and massive intracerebral hemorrhages are described in detail clinically and histologically.

2. Brief notes are presented of five other cases demonstrating the presence, in addition to superficial cortical lacerations, of petechial and massive hemorrhages within the deeper structures of the brain.

3. The view is expressed that the deeper forms of hemorrhage result, in part at least, from vasoparalytic phenomena caused by transmission of force from the site of impact to the deeper structures of the brain.

4. The changes described in this paper represent a more severe stage of brain damage than cerebral swelling and edema, described in the first paper of this series.[9]

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TREATMENT OF COMPOUND SPINE INJURIES IN FORWARD ARMY HOSPITALS

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While complete standardization in treatment of compound spine injuries is impossible and must be modified by intelligent consideration of particular problems in individual cases, it has proved desirable to make their management as uniform as possible. This has always been a difficult and discouraging group of cases to treat and perhaps for this reason they have been neglected. The necessity for rapid evacuation, often under difficult circumstances, from the scene of injury plus the problems arising from handling large numbers of casualties at one time make the care of these injuries in military surgery an even more arduous task than in civilian practice.

Although optimum methods of treatment are still uncertain, at least many common errors, both of commission and omission, are avoidable. Astute medical judgement, skillful surgery, and most important of all, meticulous nursing care are all essential to adequate treatment in these cases. Observation of large numbers of spine injuries in forward medical installations in the European Theater of Operations between June, 1944 and February, 1945 has led to the conclusions outlined in this report.

CARE OF THE PATIENT IN THE HOSPITAL AND IN TRANSIT

When a casualty arrives at a Field or Evacuation Hospital with the diagnosis of spinal injury, or when this diagnosis becomes apparent after admission, neurological examination with careful notation of the findings should be part of the initial examination. If associated shock is present demanding immediate treatment, neurological examination should be made as soon as the patient's general condition warrants. It is most important that every patient with a wound of the neck, shoulders, thorax, abdomen, or back have sensation and voluntary motion of the extremities briefly checked to rule out the possibility of a spinal injury. This is particularly true of penetrating chest and abdominal wounds, where too often preoccupation with these lesions has resulted in unfortunate neglect of a spinal injury.

All of these patients should have an indwelling urethral catheter inserted under aseptic precautions at the earliest possible moment to obviate urinary retention and overflow incontinence. If marked distention is once permitted, subsequent recovery of bladder tone is greatly delayed. Repeated catheterizations must be avoided. Considerable care should be taken to use a sufficiently large catheter to prevent leakage, and to secure the catheter firmly in position. The drainage tubing should always be attached to the litter or cot in such a manner as to relieve any possibility of tension. Personal super-

vision of these details by medical officers is necessary to keep catheters from leaking and coming out unnecessarily. Indwelling urethral catheters should be irrigated at least once daily and changed weekly. Suprapubic cystostomy is not indicated in forward hospitals except in the presence of a purulent urethritis or some other local contraindication to an indwelling catheter. Tidal drainage has proved entirely impractical in forward units, where equipment, time, and trained personnel are all limited.

The importance of the earliest possible institution of measures for care of skin with impaired nerve supply cannot be overemphasized. The sequelae of severe pressure necrosis over bony prominences are so distressing that all possible measures of prevention, no matter how laborious or how time-consuming, should be attempted. As soon after admission to the hospital as possible, the patient should be placed on a "mattress" made of at least three folded dry blankets placed on the litter. This "mattress" should be covered at all times by a dry clean sheet stretched tightly and kept as free from wrinkles as possible. Support is placed at once under the calves so that the heels do not touch the bed at any time. It is unwise to place additional support under the sacral region, as is sometimes practised, unless an inflated rubber ring is available.

The care of the skin in the entire area below the level of the lesion, with especial attention to the sacrum, trochanters, and heels, is highly important. This entire area (not just the back) should be washed with soap and water, lightly massaged, carefully dried and powdered at least once and preferably twice daily. The patient's position on the litter or cot should be adjusted at least every 4 hours day and night. This does not necessarily mean moving from back to abdomen or even from side to side. The buttocks can be raised alternately 2 to 3 inches by the use of small soft pillows or folded sheets or towels. The legs can be shifted a few inches or rotated a few degrees. They should always be kept separated. This type of adjustment can be carried out easily and safely by one attendant. Wet sheets or blankets demand immediate replacement. No adhesive tape is permissible on the anesthetic skin of back or legs. All of the foregoing measures are feasible in forward units except under the most extenuating circumstances.

If operation in a forward hospital is not contemplated, evacuation to a neurosurgical center must be expedited. The patient should never be evacuated, however, until the preparations outlined above have been carried out. That is, he should be lying on a "mattress" of blankets covered with a dry sheet, the heels should not be touching, an indwelling catheter should be in place and arrangements made so that it is never clamped off in transit. A small support, such as a folded towel or sheet, should be placed under the spine at the point of injury if it is in the lumbar region. If the cervical spine is involved, the shoulders should be raised 2–4 inches so as to allow the head to fall back slightly, and sand-bags, shoes, or a blanket-roll placed beside the head to prevent lateral deviation or rotation. The prone position is satisfactory often for short moves except in cervical lesions.

INDICATIONS FOR EARLY LAMINECTOMY

Perhaps the most difficult and controversial problem for the surgeon is the determination of which injuries due to gun-shot and shell-fragment wounds of the spine should be subjected to early surgery. Those that warrant laminectomy in forward units (i.e., Field Hospitals, Evacuation Hospitals, and General Hospitals in the Communication Zone with a 10-day holding policy) should be limited to the following groups of cases. It will be found that these constitute about 40 to 50 per cent of the total number of spine injuries seen. All others should be evacuated at once to neurosurgical centers in the rear for further care.

1. Patients who exhibit a neurological picture consistent with an incomplete transection of the spinal cord where this picture has altered since the time of injury, especially where the neurological deficit is increasing. These cases have been extremely rare.

2. Patients with a neurological picture of incomplete cord transection where X-rays reveal clear-cut encroachment on the spinal canal by a foreign body or reveal definite fracture of a lamina or pedicle with or without depressed bone fragments. If the foreign body lies on the same side of the spinal cord by X-ray as the wound of entrance, the prospects should be considered much more favorable than if it has crossed the midline.

3. Patients with a neurological picture of incomplete cord transection or of a cauda equina lesion who have associated thoracic or abdominal wounds requiring surgery and therefore making transportation undesirable for 10–14 days. In these cases it is frequently possible to perform laminectomy under local anesthesia within 48–72 hours after the chest or abdominal operation has been carried out, and certainly within a week.

4. Patients who have sizable wounds directly over or near the spinal column and are leaking spinal fluid profusely. If such a gross leak persists it is usually due to a spicule of bone or to a foreign body itself protruding through the dura and thus maintaining the opening. Adequate debridement of such a wound includes removal of comminuted and depressed bone fragments, following which the fluid leak is stopped by closure of the dura, if possible, or by careful approximation of muscle across the bony defect. Small wounds with only slight leakage of spinal fluid, especially if not directly over the spine, will usually seal themselves after debridement and may be safely watched.

When neurological examination reveals a complete physiological transection of the cord at a well-demarcated level and there is a history of instantaneous loss of all motion and sensation from the moment of injury with no subsequent change, then early operation is not indicated. If X-rays demonstrate that a foreign body has traversed the spinal canal or actually lies across the canal, or if bony fragments largely obliterate the canal in instances of complete physiological transection, the outlook is even more hopeless.

In the presence of either complete or incomplete physiological transection of the cord, laminectomy is not indicated in the absence of X-ray

evidence of fracture of the vertebrae or of a foreign body encroaching upon the spinal canal. This, of course, implies satisfactory X-rays, which may require some perseverance. Spinal shock as a result of trauma in the neighborhood of the spinal column without actual mechanical pressure on the cord or division of its continuity is a definite entity. Decompression of such a concussed cord by removal of normal laminae is felt to be of no avail. Any improvement which may follow such surgery probably would have occurred anyway in about the same period of time.

Most, if not all, spinal injuries involving the cauda equina should be explored. However, there is not the urgency for laminectomy here (below L-1) that there is at higher levels, since presumably in the nerve roots of the cauda regeneration after injury will take place and nerve suture may be possible. Therefore, unless the patient must be kept in a forward area because of associated injuries he should be evacuated as quickly as possible to a neurosurgical center for this procedure.

COMPLETE *VS.* INCOMPLETE TRANSECTION

The foregoing criteria for early surgery make it paramount that the difference between complete and incomplete physiological transection of the cord be appreciated as accurately as possible. To this end, the patient must be both questioned and examined with great care.

Except in high cervical lesions, these patients usually remain conscious and alert from the moment of injury and can give an account of their course with great accuracy. If there was abrupt and complete loss of motion and all forms of sensation from the instant of injury ("dead all over") with no subsequent change, the outlook is almost certainly hopeless. If, however, the patient has at any time experienced pain in the legs, sensations of warmth or cold, or paresthesias of any kind, or if he has been able to pass any urine spontaneously, the outlook should be considered much more favorable.

Clinical examination is more reliable. In complete transection of the cord there is a well-demarcated level of anesthesia which corresponds to the known anatomical segmental configuration. This level remains consistent. There may or may not be a band of hyperesthesia above this level. All forms of sensation are lost. It is important to test for light touch, pain, position sense, and especially deep pressure sense over the calves and soles of the feet. In total transections, flaccid paralysis is complete below the segment of the lesion and areflexia is present.* Urinary retention occurs. Priapism and vasomotor changes may be present but are unreliable criteria.

On the other hand, if any appreciation of touch, pain, position, or deep pressure is present on either side below the level of injury to the cord, or if any reflex activity is present, or if there is any muscle tone or voluntary movement, the lesion must at least tentatively be considered an incomplete one. An accurate knowledge of the segmental level of muscle innervation is,

* Occasionally, immediately after injury, a slight sluggish dorsiflexion in response to plantar stimulation is found bilaterally.

of course, essential. In incomplete or suspected incomplete lesions, repeated neurological examination by the same examiner at frequent intervals together with consideration of the X-ray findings will determine disposition.

An incomplete lesion of the cord proper (cervical and thoracic spine) will not be confused with a complete but asymmetrical lesion of the cauda equina (lumbar and sacral spine) if it is remembered that the conus medullaris lies opposite D-12 and L-1. That is, there may be complete loss of sensation and motion below say L-2 on one side and below L-5 or S-1 on the other, which is in no sense an incomplete lesion.

Lumbar puncture and estimation of spinal fluid dynamics (Queckenstedt test) have been of no particular value in compound lesions of the spine in differentiating complete from incomplete transections, or in estimating suitability for operation. A foreign body or depressed laminar fragment may be present within the spinal canal producing local involvement of the long tracts of the cord in the presence of a normal rise and fall of spinal fluid pressure on jugular compression and also in the presence of clear spinal fluid. When there has been dural penetration with a spinal fluid leak, manometry becomes unsatisfactory, and this is the rule. Blood in the spinal fluid at lumbar puncture does not necessarily mean dural penetration as it may be found in simple concussion. Decision, therefore, as to operation is more intelligently made on the basis of the clinical criteria already discussed, and spinal fluid studies have been largely abandoned in forward hospitals.

LAMINECTOMY

Laminectomy is most satisfactorily carried out under local 1 per cent procaine anesthesia. Pre-medication with morphine sulphate is practised except in lesions of the cervical cord or whenever there is involvement of the accessory muscles of respiration, in which case it should be strictly avoided. Whether general or local anesthesia is used, special attention should be given to support the upper chest from the operating table so that respiratory excursion is not embarrassed. Constant bladder drainage is uninterrupted throughout operation.

Cases that are suitable for laminectomy usually do not involve more than one or two vertebrae and consequently a large exposure is not necessary. Stripping the muscles from the vertebral spines and laminae is the most shocking part of this operation and should be carried out on only one vertebra above and below the involved segments in most instances.

The most important step of the procedure is the complete removal of all bone fragments and foreign bodies that encroach upon the spinal canal. Laminectomy can usually be started at the site of the traumatic defect. If this defect is small, it may be wiser to start the removal of bone at an uninjured point. Wide decompression by removal of additional laminae that have not been fractured is unnecessary. The operator must be certain that displacement of the dural envelope from every aspect is relieved. Extradural blood clot should be thoroughly evacuated.

If the dura has been penetrated, exploration within is carried out and a small catheter passed upward and downward to make certain there is no unexposed encroachment on the cord. Operative manipulation of the cord proper is of no therapeutic value and should be avoided. The dura is closed with fine silk sutures if possible or with a fascial graft.

If the dura has not been penetrated, a tiny opening is usually made opposite the point of dural compression. This is of no therapeutic value but of considerable prognostic importance to subsequent management of the patient. The condition of the cord, the character of the spinal fluid, and the presence of obstruction to the passage of a small catheter are ascertained and recorded.

The human fibrin foam and thrombin now available are especially useful in controlling bleeding from epidural vessels and from fractured vertebral bodies and pedicles.

Unless gross sepsis is present, these wounds should be closed in layers without drainage, muscle being carefully approximated in the midline in the region of the bony defect. Penicillin and sulfanilamide powder are used locally. Wounds of entry or exit, unless exactly in the midline, are debrided separately. If the dura has been left open, one must be particularly careful to close at least the muscular layers of these separate wounds to prevent spinal fluid leakage.

POSTOPERATIVE CARE

After operation the patient is kept flat on his back from 12–24 hours unless vomiting, and from then on his position is changed every 2–4 hours. Care of the skin, with protection of bony prominences against pressure necrosis as previously outlined, is resumed immediately. The patient is transferred to a regular mattress, or air mattress if available, placed on a cot or firm bed. Constant bladder drainage should be uninterrupted. An enema every second day is administered as soon as the patient begins to eat.

These patients may be evacuated then to a neurosurgical center in 3–4 days if there are no complications, or otherwise whenever associated injuries permit. All precautions outlined for the transportation of a patient not operated upon should apply with equal rigidity to the patient who has undergone operation. Every effort should be made to expedite evacuation and prevent delays in transit.

THORACOLUMBAR SYMPATHECTOMIES EXAMINED WITH THE ELECTRICAL SKIN RESISTANCE METHOD*

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IT HAS been found that areas of skin denervated by sympathectomy can be quickly and accurately outlined by means of the electrical skin resistance method, using a small portable dermometer. Thus far this method has been used only for upper thoracic and lumbar sympathectomies, both of them performed most frequently in the treatment of peripheral vascular diseases.[5,7] In this study it was used on thoracolumbar sympathectomies (removal of ganglia from T-9 to L-1 inclusive) performed in the treatment of hypertension.

In the previous studies it was found that the electrical skin resistance method made it possible to follow the distribution of sympathetic nerves far beyond where they have been traced by anatomical dissecting methods. Thus, it was demonstrated that the sympathetic nerves are distributed in regular segmental dermatomes, both on the head and on the lower part of the trunk and legs.[6,7]

The knowledge of the distribution of the sympathetic nerves as obtained from skin resistance measurements has been used clinically by neurosurgeons (1) preoperatively, to decide which ganglia must be removed to denervate certain affected areas of skin and (2) postoperatively, to determine whether the affected areas of skin have actually been denervated, that is, whether the neurosurgeons actually removed all of the ganglia that they had intended to.

In the present study we used this method in the examination of thoracolumbar sympathectomies to determine: (1) the extent of the areas of skin that are denervated by the operation; (2) how these areas agree with our present knowledge of the distribution of the sympathetic and sensory nerves in this part of the body; (3) whether the denervated areas can be used to indicate the surgical and clinical success of the operation; and, finally (4) whether regeneration of the sympathetic nerves can be detected by this method.

METHODS

For the full understanding of the use of the electrical skin resistance method it is important to know: (1) that the resistance offered to the passage

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of a minute, imperceptible current through the body is localized almost entirely in the skin; and (2) that this resistance is controlled largely through the sympathetic nervous system; thus removal of the sympathetic nervous system or sympathetic paralysis greatly increases skin resistance, while stimulation of the sympathetic nerves greatly decreases skin resistance.

The small portable dermometer used for these experiments has been described in detail in previous papers.[7,8] It will suffice to state here that it consists of a microammeter, a 4½-volt battery, a 1000-ohm potential divider, a telephone socket and plug, and two electrodes and connecting wires. One electrode, made of a strip of silver-plated phosphor-bronze, has the shape of a clip. By means of a specially devised clasp it can be firmly fastened to the lobe of the ear in such a way that the disc of the electrode rests directly over the front surface of the ear lobe. A paste made of kaolin and saturated zinc sulphate solution establishes contact between the electrode and the skin. A puncture made previously through the skin with a fine hypodermic needle eliminates the resistance of the skin under this electrode. This electrode remains fixed to the ear throughout the examination. The other electrode consists of a small silver-plated phosphor-bronze disc which is built into the end of an insulated wooden handle. This electrode is held in the hand of the examiner, thus making it possible to bring its flat, smooth surface into contact with the skin on any part of the body. No paste is used with this electrode. As a result of the elimination of the resistance under the ear electrode by the skin puncture, all of the remaining resistance offered by the body is localized in the skin under this movable electrode.

With one electrode attached to the ear lobe and the other in contact with the skin, the amount of current from the battery that flows through the circuit is indicated by the excursion of the needle of the microammeter. The extent of the excursion varies directly with the resistance offered by the skin under the hand electrode. When this resistance is high the needle will not move, indicating that no current is flowing, while when it is low the needle will move quickly entirely across the dial, indicating that a large amount of current is flowing. The instrument can be adjusted so that when the hand electrode is placed over a normally innervated area of skin the needle will move sharply and entirely across the dial, while when it is placed over a denervated area it will not move at all.

The sharpness of the outlines of the denervated areas depends on the difference in level that exists between the resistance of the denervated areas and the surrounding areas of normally innervated skin. Factors that promote sweating reduce the resistance on the normally innervated skin but have little or no effect on parts that have been denervated. Thus, to sharpen the outlines, the patients may be heated for a few minutes in a hot-air cabinet or under a heat cradle. Hypertensive patients rarely require much heating since in their weakened condition even the slight effort involved in sitting up in bed is apt to produce sweating on all normally innervated areas. Hence, many patients can be adequately examined in their beds on the wards. In all instances, however, in which diffuse or doubtful outlines are obtained the patients must be heated in a heat cabinet or under a heat cradle for a few minutes. Because the patients are frequently in a weakened condition for the first few days after the operation, the examinations are not usually made before the 6th to 10th postoperative day.

The mapping of the areas that have been denervated by thoracolumbar sympathectomy involves the following steps:

(1) The patient is placed in a hot-air cabinet and is heated for a few minutes. Whenever possible the areas of high electrical skin resistance are outlined before the patient leaves the cabinet. This reduces the complications in the records that may arise from sudden cooling. If the patient is too weak to sit up in the cabinet, he is allowed to lie down in bed and is then kept as warm as possible with blankets.

(2) The fixed electrode is attached to the ear lobe, and the dermometer is placed so that the dial of the microammeter is readily visible at all times.

(3) The full current from the battery is turned on. This is done by turning the knob of the potential divider as far in the clockwise direction as possible.

(4) The flat surface of the hand electrode is held against any normally innervated area of skin such, for instance, as is found on the hands and chest, and a close watch is kept on the needle of the microammeter. If in each instance the needle moves quickly across the dial it is known that the patient has been properly heated for the test. If, however, it gives only a small or no response, the patient must be heated for a longer time.

(5) The hand electrode is then touched to the skin in the region near the umbilicus, which probably has been denervated. As soon as a place is found from which no response of the needle on the microammeter is obtained, it is known that a denervated area has been located.

(6) The electrode is then allowed to slide along the skin up the trunk, that is, toward a normally innervated area, taking care to keep all of the flat surface in close contact with the skin. The location of the leading edge of the electrode where the pointer on the microammeter gives its first small flicker is marked on the skin with a black skin-pencil. This marks the upper boundary of the denervated area.

(7) This process is repeated in adjoining areas to the right and left side on the anterior surface of the trunk and the points thus obtained are joined together with a line. This line is extended as far as possible around as much of the trunk as is visible from the front.

(8) Then the electrode is moved across the denervated area down the trunk until again the needle gives the first small flicker. This point is marked on the skin and the process is repeated until the lower border of the denervated area is determined for the anterior surface.

(9) The patient is then turned around and the process is repeated until the lines are determined that mark the upper and lower borders of the denervated area on the back.

(10) The lines are then copied on body charts which show the front, rear and side views of the trunk and photographs are made of the same views.

(11) In some patients when the movable electrode is allowed to slide over the skin beyond the line that produces the first flicker of the needle a second line is found at which the response of the needle may change suddenly from 10 to 15 divisions to a sharp response entirely across the dial. The area enclosed between the two lines is called an intermediate area (in the starch-iodine-sweating test this intermediate area shows a stippling with black dots rather than a solid black coloring).

(12) Whenever intercostal nerves have been cut the trunk is examined for areas of anesthesia or dullness.

(13) After the outlines of the areas of high skin resistance around the waist have been determined, the normal areas above and below should be quickly tested again to make certain that no other areas of high skin resistance have been overlooked.

(14) To determine the location of the areas of high electrical skin resistance with relation to the spinal vertebrae, fly-screen wire cloth is first placed over as much of the area of high skin resistance as is visible on the front of the trunk and the outlines are traced on the cloth with white chalk. The patterns are then cut out with a pair of metal shears and the cut edges are bound with adhesive tape. The same process is repeated for the areas on the back of the trunk. Then the two wire patterns are fitted over the skin patterns and are held in place with adhesive tape and the patients are taken to the X-ray Department to be photographed. A single x-ray photograph usually suffices to show the wire-cloth patterns, both on the front and back of the trunk, and the dural clips which have been placed on the white rami of each of the excised ganglia. Soft iron wire, bent to conform to the outlines of the skin resistance patterns, may be used in place of the wire-screen cloth.

We were able to obtain x-ray photographs for only 16 of the 48 operations. War restrictions on photograph film prevented us from getting photographs of all of the operations.*

Twenty-four patients, with a total of 48 operations, are included in this report.

The surgical procedure used closely followed the technique described by White and

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* The staff of the X-ray Department gave us excellent cooperation in this work.

Smithwick.[9] Operations were done in two stages, usually ten days to two weeks apart. An incision was made along the lateral border of the sacrospinalis muscle, starting at the level of the 10th rib and curving outward in the lumbar region. Approximately 1½ to 2 inches of the 11th and 12th ribs were resected. The 11th intercostal nerve, muscle and vessels were severed. In a few patients the 12th intercostal nerve was cut also.

The pleura was dissected away from the vertebral bodies until, deep inside, the greater splanchnic trunk was exposed. The nerve was severed as high up in the chest as could be reached, usually at the level of the 9th vertebra. It was then followed caudally and cut off at its point of entry into the coeliac ganglion.

The sympathetic chain was then isolated and as many thoracic ganglia as could be reached were excised. A silver clip was placed at the site of each white ramus communicans, thus

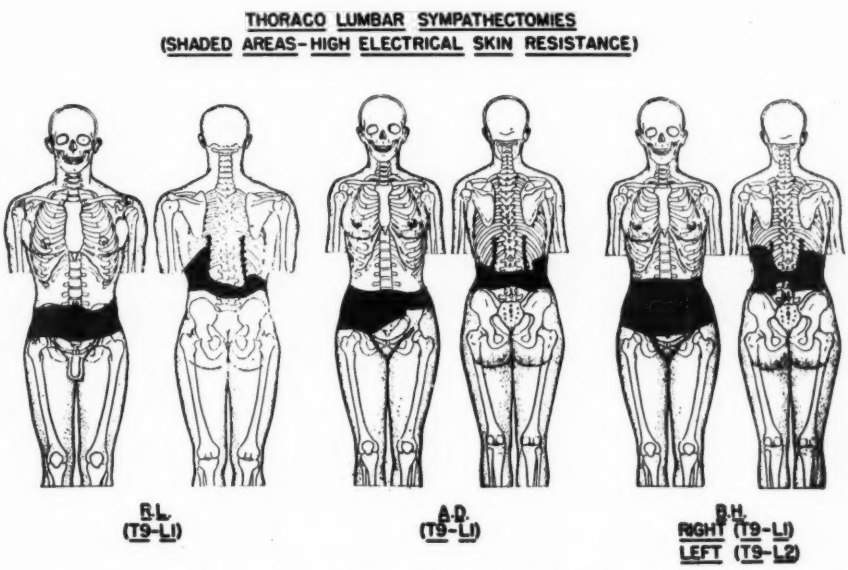

FIG. 1. Front and rear views of the trunk for 3 patients, showing the areas of high electrical skin resistance (black shading) which were produced by thoracolumbar sympathectomies. In these patients the band areas completely surrounded the trunk. The crossed black lines on the back indicate the location of the scars.

making it possible to check by x-ray the number of ganglia taken out. The lesser splanchnics were also severed.

Going below the diaphragm the lumbar trunk was isolated and the first lumbar ganglion removed. In a few cases an attempt was made to excise the second ganglion also. Silver clips were used as before.

In the great majority of cases, the surgeons stated that they had removed the sympathetic ganglia from T-9 to L-1 inclusive.

RESULTS

Despite the fact that in most of the 48 operations the surgeons stated that the same ganglia had been removed, the patterns of the areas of high

electrical skin resistance showed very great variations, not only from patient to patient, but also from one side to the other of the same patient. In each case, the area formed either a continuous band around the body, or a broken band, leaving a gap in back or in front which was always located to one or both sides of the midline. Because of these variations it is not possible to present one or two records that would be typical of the others. The 8 records in Figs. 1, 2, and 3 will give some idea of the variations in the size and shape of the patterns of the denervated areas.

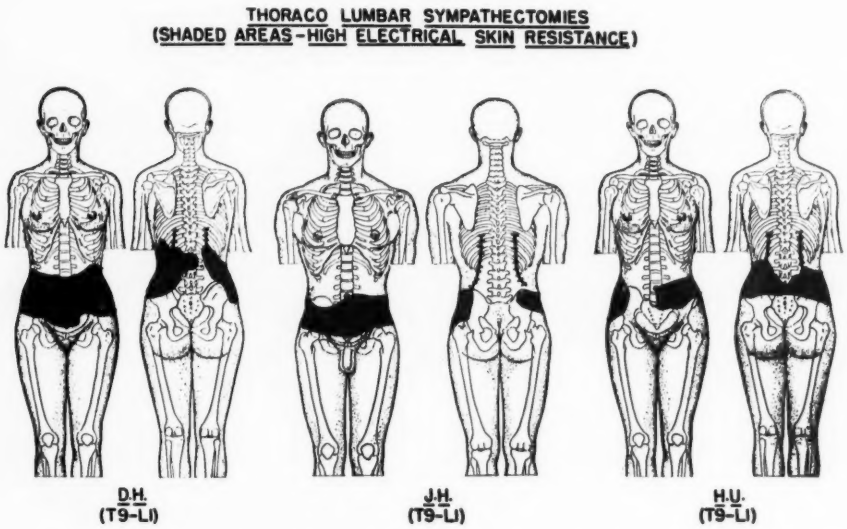

FIG. 2. Front and rear views of the trunk for 3 patients, showing band areas of high electrical skin resistance (black shading) which were produced by thoracolumbar sympathectomies. In these patients the band areas did not completely surround the trunk. A gap of normally low resistance separated the two ends of the band area.

In Fig. 1, the first record (R. L.) shows the area of high electrical skin resistance of a patient from whom the surgeon stated that he had removed the ganglia on both sides from T-9 to L-1 inclusive. The area formed a continuous band around the trunk, but was much narrower and more irregular on the posterior surface. Incidental sectioning of cutaneous nerves in making the incision probably accounts for the areas of high resistance along the outer surface of the incision on the left side.

In Fig. 1, the second record (A. D.) shows the areas of high electrical skin resistance in a patient with the same operation. This denervated area also formed a continuous band around the body. In front it was over twice as wide on the right side as on the left, and in back it was fairly evenly narrow on both sides.

In Fig. 1, the third record (B. H.) shows the area of high skin resistance in a patient from whom the surgeon stated that he had removed the ganglia from T-9 to L-1 inclusive on the right side and from T-9 to L-2 inclusive on the left side. The area formed a continuous band which was much wider in front than were either of the areas shown in the previous records, and its borders were more regular. In the middle region of the back it narrowed suddenly to a very thin strip.

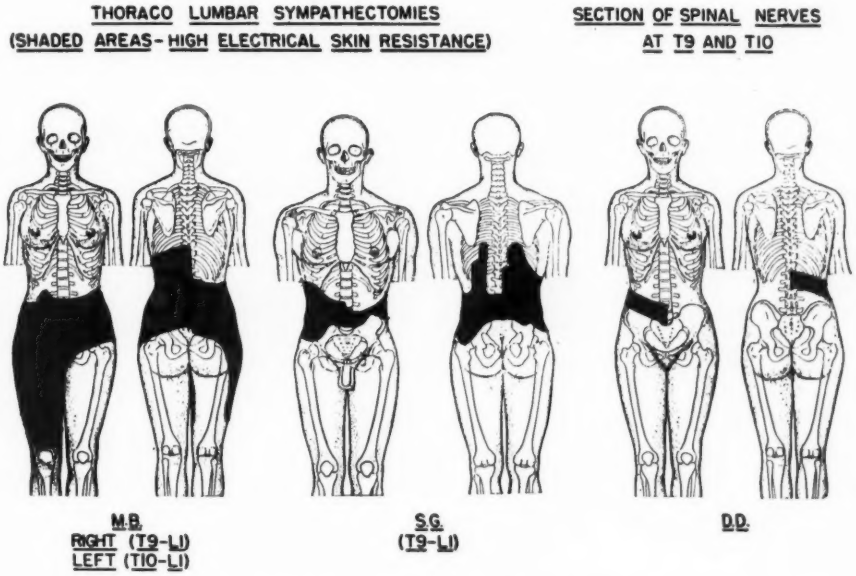

FIG. 3. Front and rear views of the trunk in 2 patients (M. B. and S. G.), showing unusual patterns of high electrical skin resistance (black shading) which were produced by thoracolumbar sympathectomies; and of a 3rd patient (D. D.) showing regular zonal pattern of high electrical skin resistance produced by a unilateral section intraspinally of the 9th and 10th thoracic nerves.

In Fig. 2, the first record (D. H.) shows the denervated area in a patient who had the usual operation (removal of the ganglia from T-9 to L-1 inclusive). In this patient the band was not continuous around the body. A small gap about one inch wide, slightly off the midline in back, separated the two ends of the band.

In Fig. 2, the second record (J. H.) shows the denervated areas in a patient who also had the usual operation. In this patient a much larger gap on both sides of the midline in back separated the two ends of the band. Otherwise the borders were even and regular.

In Fig. 2, the third record (H. U.) shows the denervated area in another patient who also had the usual operation. A gap located in front on one side

of the midline separated the two ends of the band. In back the band was almost divided again.

In Fig. 3, the first record (M. B.) shows the denervated area in a patient from whom the surgeon reported that he had removed only the ganglia T-9 to L-1 inclusive on the right side and from T-10 to L-1 inclusive on the left side. The pattern of the area is unusual in that it extended down over the anterior surface of the leg on the right side and was much wider in back on both sides than any of the patterns seen in all the other patients. A similar pattern was found in only one other of the 48 operations. It is likely that in this patient the surgeon removed some of the chain on the right side below the 1st lumbar ganglion, probably the usually fused 2nd and 3rd ganglia.

In Fig. 3, the second record (S. G.) shows the denervated area in a patient who had the usual thoracolumbar operation. This record is presented largely because the area of high electrical skin resistance extended far up on the back and included the skin entirely on both sides of the two incisions. Such areas of high skin resistance surrounded only a few of the incisions. Why they did not surround more of them we do not know.

Of the 48 operations, 11 showed partial gaps on the back and 5 on the front; 16 showed complete gaps on the back, and 2 on the front. In no case were complete gaps both on the back and on the front observed simultaneously.

An effort was made to locate the borders of the denervated areas with relation to the surface markings and the spinal vertebrae. For this purpose we used only borders that were fairly smooth and seemed to parallel outlines of regular thoracic segments. Smooth borders were found much more frequently on the anterior than on the posterior surfaces of the body. In most of these patterns in front the upper borders ran through or just above the umbilicus, while the lower borders ran through or just above the pubic arch; in back the upper borders ran through the 1st or 2nd lumbar vertebra and the lower borders through a line that was located approximately one to two inches above the level of the iliac crests.

The x-ray photographs of the wire-screen patterns that were obtained for the 16 operations showed that in most instances, in front, the upper border of the denervated area passed through the middle of the 4th lumbar vertebra, and the lower border passed through or just above the pubic arch; while, in back, the upper border passed between the 1st and 2nd lumbar vertebrae, and the lower border through the 4th or 5th lumbar vertebra.

X-ray photographs of the patterns made it possible to determine, in most instances, that the borders of the denervated areas in front were located 3 to $3\frac{1}{2}$ vertebral spaces below the analogous borders of the patterns on the back of the body. This relation may be seen also in the record obtained from a patient with a ruptured disc at T-10 in whom the 9th and 10th intercostal nerves on the right side had been sectioned intraspinally because of the presence of pain which did not seem to be fully explained by the pressure from the disc. The skin resistance pattern produced by this operation is

presented in Fig. 3 (D. D.). The area of high skin resistance produced by this operation was approximately 2 spinal segments in width, had very regular borders, and extended from the midline in front to the midline in back. In front, the upper border ran through the umbilicus and in back near the

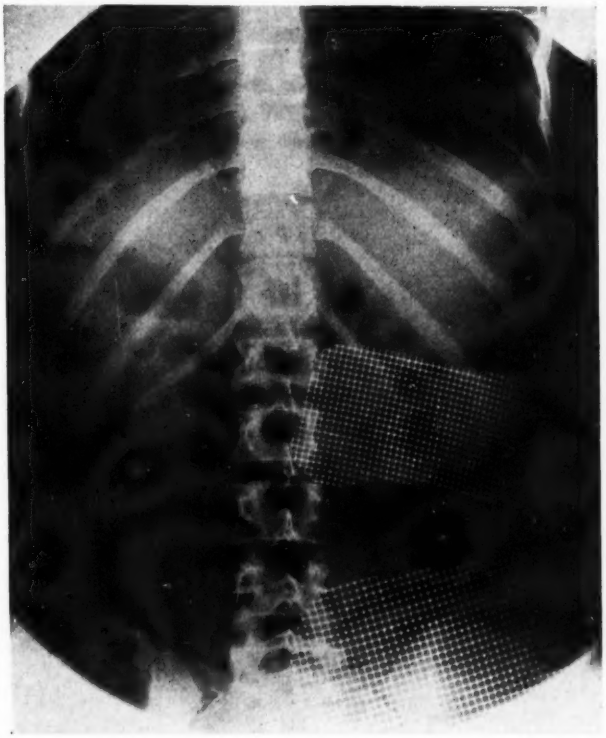

FIG. 4. X-ray photograph, showing spinal vertebrae and the wire-screen cloth cut out to conform to the area of high electrical skin resistance produced by the intraspinal section of the 9th and 10th thoracic nerves in the patient (D. D.) whose skin resistance record is presented in Fig. 3. This photograph also shows a dural clip placed intraspinally at the site of the 10th thoracic nerve. One piece of wire cloth covered the entire pattern on both the anterior and posterior surfaces of the body. The photograph thus shows the screen as it started from the midline on the back opposite the 1st and 2nd lumbar vertebrae and curved down around the body, ending on the midline in front opposite the 5th lumbar and 1st sacral vertebrae.

top of the first lumbar vertebra. The x-ray photograph of this pattern is shown in Fig. 4. It shows the wire screen as it winds around the body from the midline in back down to the midline in front. It will be seen that the upper border on the posterior surface is approximately $3\frac{1}{2}$ spinal spaces above the analogous border on the anterior surface.

The patterns of the denervated areas appeared to remain very constant from day to day. More than half of the patients were tested on several different days during the immediate postoperative period and a few patients were tested at infrequent intervals as long as six months postoperatively. On these repeated examinations the patterns showed only minimum variations, which probably resulted from technical errors. In a few instances the patterns obtained while the patients rested in bed without heating and those obtained after the patients had been heated were not identical. Even then, however, the variations were not great.

Further proof that the band areas of high electrical skin resistance had been sympathectomized, while the gaps still retained their sympathetic in-

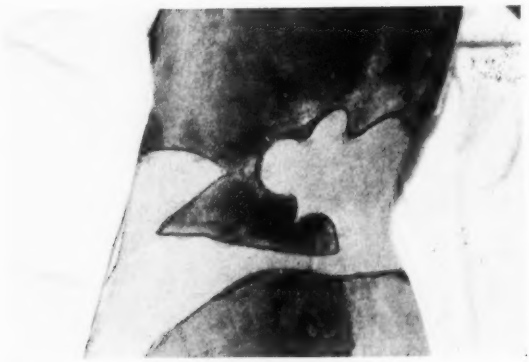

FIG. 5. Photograph showing skin resistance and sweating patterns on the back of a patient who had had a bilateral thoracolumbar sympathectomy at T-9 to L-1 inclusive. It shows the close relationship between the areas of high electrical skin resistance (bounded by black lines) and the areas that did not sweat (white), as demonstrated by the Minor starch-iodine-sweating test. The photograph shows that the partial gap in the area of high electrical skin resistance still manifested active sweating (black).

nervation, was obtained by means of the Minor starch-iodine-sweating test. The results of this test showed that the band areas of high skin resistance no longer sweated, while the gaps in the band still showed normal sweat gland activity. Fig. 5, which shows the rear view of one of the patients, gives the combined results of the electrical skin resistance and Minor starch-iodine tests and gives a typical demonstration of this relationship. The following procedure was used in obtaining this record. The patient was heated and then the area of high electrical skin resistance was outlined on the skin with a black skin-pencil. Shortly after the patient cooled off, the skin was painted with the standard mixture of alcohol, castor oil and iodine, and then dusted with rice-starch powder. Then the patient was heated again until he sweated actively on all normally innervated parts of the body. These areas of skin turned black. It can be clearly seen that the area of high electrical skin re-

sistance enclosed between the black lines did not sweat while the surrounding areas and the gap showed active sweating.

In patients who have a large area of intermediately high skin resistance the heavy black coloring coincides closely with the outer boundary of this area, while the intermediate area shows only a scattering of black dots.

Only toward the end of the present survey did it become clear that the section of the intercostal nerves might affect not only the skin resistance patterns but also the clinical results of the operations. All of the patients are now being reexamined for areas of dullness or anesthesia. In general it can be said that the feeling, so often experienced by patients, that a wire is being drawn tightly around their waist is localized very closely to the narrow area of dullness or anesthesia.*

REGENERATION OF SYMPATHETIC NERVES

About half of the patients have been reexamined several months to one year postoperatively. Thus far, it has been observed that the areas of high skin resistance become greatly reduced in size. They are narrower and the gaps are more frequent and wider. The changes in patterns agree closely with the results of the Minor starch-iodine-sweating tests. The results of these observations on regeneration patterns will be reported when an adequate time has elapsed for correlation with the clinical effects. The areas of anesthesia and dullness produced by the section of the 11th and, occasionally, the 12th intercostal nerves, also undergo marked changes.

POSSIBLE FACTORS INVOLVED IN THE PRODUCTION OF THE IRREGULARITIES AND GAPS IN THE DENERVATED AREAS

The first and most obvious explanation of the narrowness of the patterns is that the surgeons had not actually removed as many as five ganglia on both sides. In each of the seven patients with dural clips and x-ray photographs, five dural clips were found on both sides of the spinal cord. The five clips, which had been placed at the sites of each of the removed ganglia, were located in most instances opposite each one of the spinal vertebrae, usually from T-10 to L-2 inclusive. It is likely that in most of the other patients also five ganglia had been removed.

The narrowness of the denervated areas may be explained, at least in part, by the phenomenon of dermatome overlap. Thus, the area denervated by sympathectomy from T-9 to L-1 inclusive would be narrowed at the top by the overlap from the next higher intact dermatome, namely T-8, and it would be narrowed at the bottom by the overlap from the next lower intact dermatome, namely L-2. Thus, it is probable that the denervated areas included only three or four dermatomes rather than the full five.

* Blades and Dugan ("War wounds of the chest." *J. thorac. Surg.*, 1944, *13:* 294–306) have recently observed in their operations on patients with war wounds of the chest that section of the intercostal nerves produces pain which cannot be satisfactorily treated. They have modified this operation to avoid, so far as possible, any injury to these nerves.

The irregularity and asymmetry of the denervated areas and the presence of gaps, or areas that did not become denervated, may be explained in large part by the irregular distribution of the various divisions of the intercostal nerves to the skin. From the work of Johnston[4] it is known that the posterior primary rami (the posterior cutaneous divisions of the intercostal nerves —see Fig. 6) which innervate the middle of the back, that is, the same part of

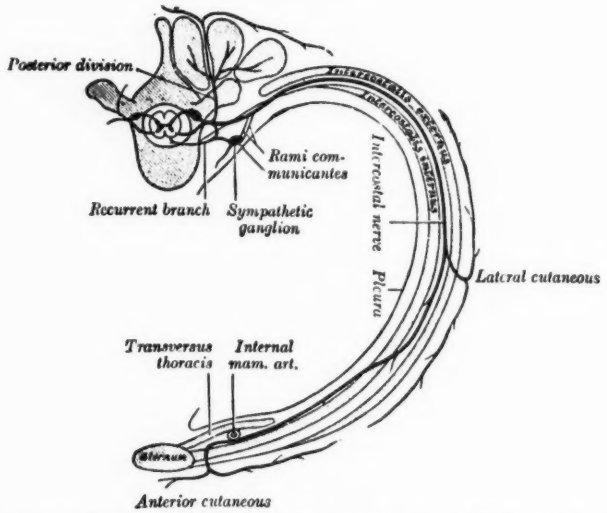

FIG. 6. Diagram taken from Gray's *Anatomy* (Philadelphia: Lea & Febiger, 1936, 23rd ed., p. 935), showing the cutaneous divisions of a typical intercostal nerve (posterior primary ramus).

the back that most frequently fails to become denervated by the thoracolumbar sympathectomies, have a very irregular distribution. These posterior rami are divided into medial and lateral branches (designated by Johnston as internal and external branches) which may innervate areas of skin that are separated by several spinal segments, and which are not symmetrically located on the two sides of the body. The drawing at the left in Fig. 7 taken from Johnston shows a dissection in one cadaver of the posterior primary rami as they emerge from the muscles and enter the skin. The drawing at the right in Fig. 7, also taken from Johnston, shows in one cadaver the distribution of the posterior primary rami to the skin. The areas innervated by the different thoracic nerves varied considerably in size and shape and location, and were not symmetrical on the two sides of the body. They did not show a regular segmental distribution. Thus, for instance, T-8 on the left side extended from the spinal level of T-11 to L-1, while on the right side it extended from L-1 down to L-3. On the right side a gap, filled in by the T-9 and T-11 areas, completely separated the T-10 areas from the midline. Five other cadavers dissected by Johnston showed equally wide variations in dis-

tribution of the posterior primary rami. Thus, it becomes clear, even from this small number of dissections, that sections of the sympathetic nerves in this region would not always denervate regular segmental patterns.

It is likely that the variations in the distribution of the anterior primary rami (anterior cutaneous divisions of the intercostal nerves) may likewise explain the presence of areas of skin near the middle on the anterior surface

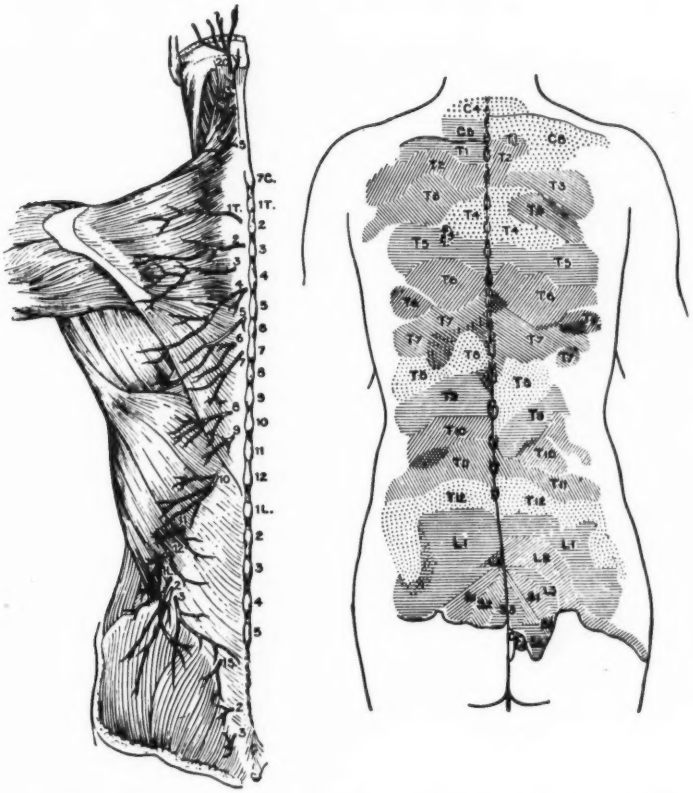

FIG. 7. Diagram taken from Johnston,[4] showing at the left, for one cadaver, the distribution of the cutaneous branches of the posterior divisions of the intercostal nerves (posterior primary rami) after they left the muscles and just before they entered the skin; and, at the right, showing the areas of skin which were found, in one cadaver, to be innervated by the posterior cutaneous divisions of the intercostal nerve.

of the trunk which still retain their normal function after the thoracolumbar sympathectomy. The distribution of these rami may show an equally wide variation. It is possible also that the lateral branches of the anterior rami may likewise manifest an irregular distribution and asymmetry.

It is of interest in this connection that the dermatome charts constructed by Head[3] show a considerable amount of variation in the shape of the thoracic dermatomes and that, in several instances, they show gaps in the individual dermatomes.

The isolated observation made on the patient with the intraspinal section of the spinal nerves at T-9 and T-10 mentioned above may throw some light on the origin of the irregularity of the patterns of the denervated areas produced by removal of the sympathetic ganglia. Figs. 3 and 4 respectively show the skin resistance and x-ray patterns of this patient. In contrast to the patterns produced by sympathectomy this pattern had extremely regular borders and followed the regular segmental outlines. This suggests that the sympathetic nerves may leave the spinal nerves in a more regular segmental distribution than they do when they leave the sympathetic ganglia.

DISCUSSION

The results of these observations have shown that in most instances the thoracolumbar sympathectomies actually denervated only a very narrow band of skin around the middle of the trunk.* Therefore, it is obvious, since the proportion of the denervated areas to the total surface area of the body was so small, that the denervation of the peripheral vascular bed cannot play any important part in the production of the great drop in blood pressure. Clearly, denervation of internal organs must account for this effect.

Clinically, because of the large variations in the width of the patterns and the presence of gaps, the electrical skin resistance mapping test can be used on thoracolumbar sympathectomies only as a rough indicator of the number of ganglia that have been removed.

At present it is not known whether or not there is a correlation between the extent of the denervated areas and the decrease in blood pressure or the permanency of the clinical effects. Only observations made over a period of several years will reveal whether or not such a correlation exists. On the basis of observations made on lumbar and upper thoracic sympathectomies, it is likely that this test will give a simple objective measure of regeneration of sympathetic nerves also in thoracolumbar sympathectomies.

The results gain interest from the new light that they have thrown on the distribution of the sympathetic nerves and of the sensory nerves to the skin in this part of the body. The great variation in the distribution of these nerves would never have been suspected except for the few anatomical observations reported by Johnston in 1908, in an important paper which has long been overlooked,[4] on the distribution of the posterior primary rami.

* Since the writing of this paper one of us (C.P.R.) has had the opportunity to examine at the Lahey Clinic in Boston a large number of patients who have had more radical thoracolumbar sympathectomies. These patients, most all of whom were operated on by Dr. James L. Poppen, had patterns which differed quite markedly from those presented in this paper, particularly in that in most all instances the skin of the lower sacral and lumbar segments of the legs showed complete denervation.

It may be noted that the outlines of the patterns produced by the upper thoracic and by the lumbar sympathectomies are much more regular than those produced by the thoracolumbar sympathectomies; also the patterns do not show any gaps. We are unable to explain these differences.

Here, again, it has been possible with the electrical skin resistance method to work out strictly anatomical problems of nerve distribution which, because of the fineness and imperceptibility of the terminal nerve branches, would not have been possible with any of the ordinary dissecting methods used in anatomical studies.

It is possible that the gaps in the patterns depend upon a sympathetic supply which reaches the skin through entirely unsuspected channels. An explanation might thus be obtained for the highly baffling observations made on hypertensive patients by Grimson[2] that sympathetic activity such as sweating recurs in the thoracic region even after the entire sympathetic chain has been removed bilaterally from one end of the body to the other; also for the observations that total sympathectomy has very little effect on dogs.[1]

SUMMARY

1. The electrical skin resistance method was used to outline sympathectomized areas of skin in 24 patients with a total of 48 thoracolumbar sympathectomies (removal of the ganglia at T-9 to L-1 inclusive).

2. In each patient the denervated area formed a narrow band which either completely or partially encircled the trunk. In many instances the outlines of the denervated areas were very irregular and asymmetrical.

3. On the anterior surface of the trunk the upper borders of the denervated area were located at the level of the umbilicus, and the lower borders at the level of the pubic arch; on the posterior surface the upper borders were located near the level of the 1st lumbar vertebra and the lower borders at the level of the 4th or 5th lumbar vertebra.

4. By means of x-ray photographs it was demonstrated that a distance of 3 to 3½ spinal segments separated the analogous borders of the denervated areas on the anterior and posterior surfaces of the body.

5. In the instances in which the sympathectomy did not denervate a band area which completely surrounded the body, the gap in the band was located near the midline, either in back or in front, and varied in width from 1 to 4 inches.

6. The band areas of high electrical skin resistance did not sweat, while the partial or total gaps showed normal sweat-gland activity.

7. Sufficient time has not yet elapsed to determine whether or not any relation exists between the characteristics of the denervated areas (their width and location, irregularity of their outlines, presence or absence of gaps) and the clinical success of the operation or the permanency of the results.

8. Since the areas of skin denervated by the sympathectomy form such a small part of the total skin area of the body it would seem that the denerva-

tion of the peripheral vascular bed cannot play an important part in the achievement of the clinical results.

9. The irregular asymmetrical distribution of the posterior and anterior primary rami of the intercostal nerves and their widespread overlap explain, at least in part, the irregular asymmetric outlines of the denervated areas as well as the presence of the gaps.

10. The results thus brought further evidence to show that the electrical skin resistance method makes it possible to determine facts regarding the distribution of nerves which ordinary anatomical dissection methods probably could not reveal.

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PERIPHERAL NERVE SURGERY—REVIEW OF INCISIONS FOR OPERATIVE EXPOSURE. PRELIMINARY REPORT*

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IN THE military service during war, injuries to peripheral nerves are amongst the most commonly observed in the wounded soldier that survives. This is easily understood when one realizes how multiple are the penetrating wounds that may be suffered by a single man, from the spraying of shell fragments. Because of the penetrating quality of, and multiplicity of fragments from highly explosive shells and land mines, nerves that are normally well-hidden and protected are just as vulnerable as are those near the surface. The purpose of this paper, therefore, is to review the most logical approaches for adequate exposure of the various nerves and to present new incisions developed on the Neuro-Surgical Service at Wakeman General Hospital.

These modifications are based on the study of 1567 peripheral nerve lesions.

In devising the new incisions, adherence to the following surgical principles was found to be of great advantage.

(1) Adequate exposure of the injured nerve.
(2) The approach to deeply lying nerves is made through fascial planes, rather than by penetrating through muscle substance.
(3) If a muscle must be penetrated, it is always split in the direction of its fibers.
(4) When a muscle must be divided, it is severed only at its point of fascial attachment, so that it may be easily repaired.
(5) Shortening of a normal long bone, or resection of a clavicle are avoided when possible.
(6) Skin incisions that are perpendicular to flexion creases must be avoided, since a traction scar will invariably result.[4] Not only is the skin involved in the production of traction scars, but subcutaneous tissues and fascial layers as well.

The incisions of the extremities have for the most part been divided into three parts. This is especially true for incisions about the cubital and popliteal fossae. One part alone is usually sufficient for repair of the nerve, while two or all three parts may be used when rerouting as well as repair is necessary.

INCISIONS IN THE UPPER EXTREMITY

The Median Nerve. The median nerve in the arm, above the elbow joint, is easily explored through part 1, Fig. 1; this consists of a longitudinal incision over the mesial surface of the arm. It lies in the plane between the bor-

* Presented at the meeting of the Texas Surgical Society, Fort Worth, Texas, April 2 to 5, 1945.

ders of the biceps and triceps muscles. The median nerve in the forearm is exposed through a longitudinal incision on the volar surface of the forearm. It is usually placed mesial to the border of the brachioradialis muscle (part 3, Fig. 1). Through this incision the pronator teres muscle is exposed and split in the direction of its fibers where deep within its substance the median nerve courses. Part 2 of the incision in Fig. 1 is a transverse incision following a flexion crease in the cubital fossa. When these incisions are united, a modi-

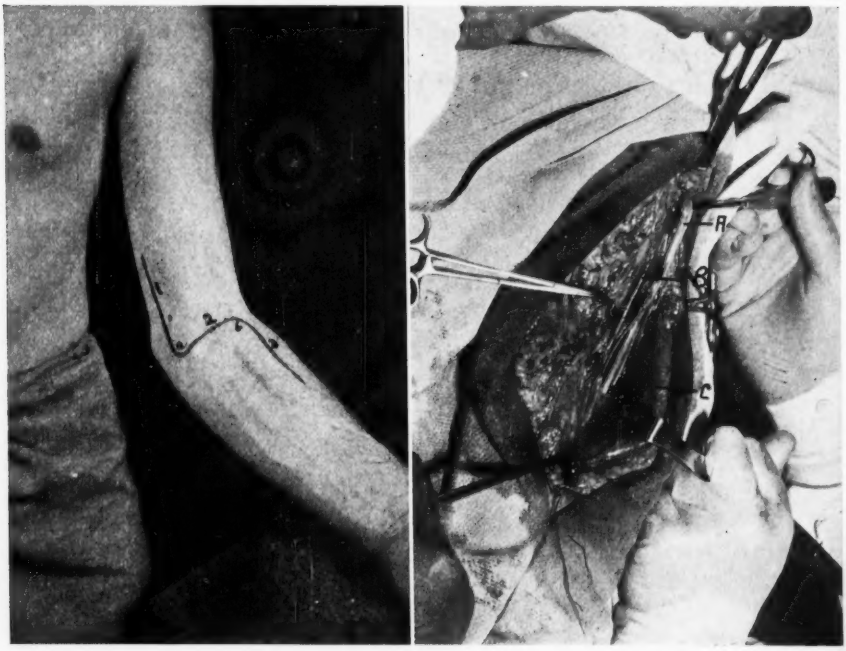

FIG. 1 (*left*). Incision for exploration of median nerve in arm and forearm.
FIG. 2 (*right*). Exposure of the median nerve in the arm and forearm accomplished by the use of the combined Z-shaped incision. (A) Median nerve. (B) Brachial artery. (C) Pronator teres muscle.

fied Z is formed, thus avoiding an incision crossing a flexion crease. The flaps, *a* and *b*, are now retracted and through this wide exposure the median nerve may be traced along its course in the arm and forearm (Fig. 2). A transplantation of this nerve may also be carried out.[1] It is possible to explore the radial and ulnar nerve as well as the median nerve through this incision. Such an incision heals without distortion and avoids the flexion contractures shown in Figs. 3 and 4.

The Ulnar Nerve. The exposure of the ulnar nerve in the arm is accomplished through the same incision as for the median nerve as shown in part 1, Fig. 5. The ulnar nerve in the forearm is explored through part 3, Fig. 5.

FIG. 3 (*left*). Traction scar resulting from an incision perpendicular to flexion crease at the cubital fossa.

FIG. 4 (*right*). Extensive incision crossing the flexion crease at the cubital fossa.

This is a longitudinal incision following the margin of the flexor carpiulnaris muscle. Part 2 of the incision in Fig. 5 is a 5 cm. curved incision over the region of the medial epicondyle. This is used to explore and transplant the

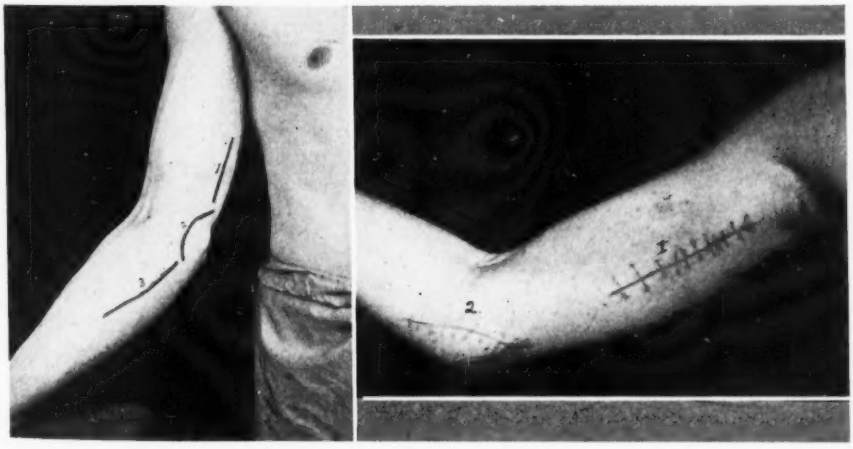

FIG. 5 (*left*). Incision for exposure of the ulnar nerve in the arm, at the ulnar notch, and in the forearm.

FIG. 6 (*right*). Incision for exposure of the ulnar nerve. Incision #1 was used for neurorrhaphy, after transplantation of nerve was performed through incision #2.

ulnar nerve to the volar surface of the forearm.[6] Parts 1, 2 and/or 3 of this incision may be used depending upon the need and location of the nerve injury as shown in Fig. 6.

The Radial Nerve. The radial nerve in the arm is exposed through part 1 of the incision in Fig. 7. This is a longitudinal or oblique incision over the lateral surface of the arm parallel to the radial groove. The radial nerve in the forearm, part 3 of Fig. 7, is exposed through a longitudinal incision over the volar surface of the forearm, being placed laterally over the border of the

FIG. 7 (*left*). Incision for exposure of the radial nerve: (1) in the arm, (2) lateral to the cubital fossa, (3) in the forearm.

FIG. 8 (*right*). Exploration of the brachial plexus. Transverse axillary incision.

brachioradialis muscle. The radial nerve at the level of the cubital fossa is best exposed by a 5 or 6 cm. semi-circular flap incision as shown in part 2, Fig. 7. This incision is made laterally over the region of the lateral epicondyle in order to avoid crossing the flexion crease. When this flap is retracted medialward, the nerve may be exposed as it lies between the biceps tendon and the brachioradialis muscle. From here it may be followed along its course beneath the brachioradialis muscle to its point of division into the deep and superficial radial nerves.

The Brachial Plexus. Transverse axillary incision. Exposure of the entire brachial plexus in the axilla can be easily accomplished by a transverse incision following a flexion crease across the entire axilla as shown in Fig. 8. This is the incision of choice, especially for exposing the radial nerve since it lies posteriorly in the axilla. Such an incision avoids the deformity and dis-

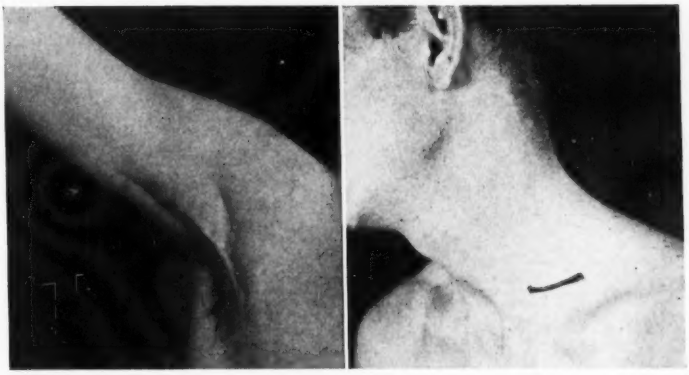

FIG. 9 (*left*). Traction scar resulting from an incision perpendicular to flexion crease at the axilla.
FIG. 10 (*right*). Exploration of the brachial plexus. Supraclavicular incision for exposure of the brachial plexus in the neck.

ability of a contraction scar which follows an incision perpendicular to the flexion creases in the axilla as shown in Fig. 9.

Supraclavicular incision. This is a 4 cm. transverse incision 2 cm. above and parallel to the clavicle as shown in Fig. 10. This incision is used to expose the brachial plexus in the neck. It is also used for exposure of the anterior scalene muscle.[9]

Infraclavicular exposure of the brachial plexus. This is the accepted approach for exposure of the brachial plexus beneath the pectoralis major

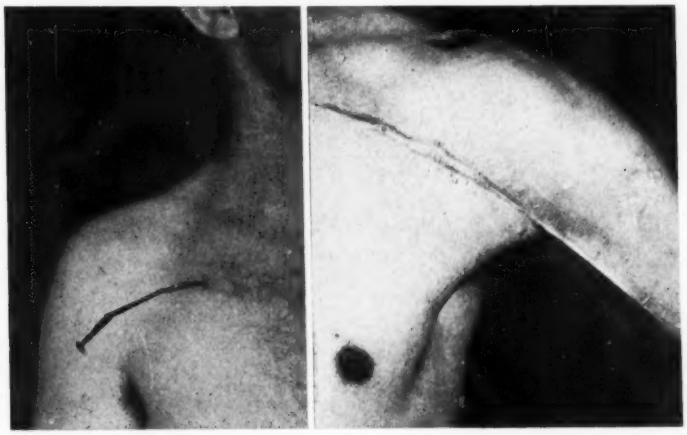

FIG. 11 (*left*). Exploration of the brachial plexus. Infraclavicular incision for exposure of the brachial plexus beneath the pectoralis major muscle.
FIG. 12 (*right*). Traction scar resulting from erroneously planned infraclavicular incision crossing the anterior axillary fold.

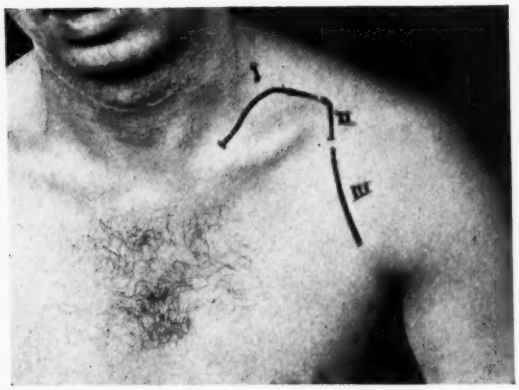

FIG. 13. Exploration of the brachial plexus. Transclavicular approach for exposure of the brachial plexus above, below, and beneath the clavicle.

muscle. It consists of a transverse incision beginning some 5 cm. below the clavicle at the level of about the middle third of the clavicle, and continuing lateralward into the arm over the region of insertion of the pectoralis major muscle, as shown in Fig. 11. The pectoral muscle is exposed and split in the direction of its fibers, bringing the roots of the brachial plexus into view. To venture into the axillary fold produces the result seen in Fig. 12.

Transclavicular approach to brachial plexus (incision of Stone).[7] By this approach the brachial plexus may be exposed above, below, and beneath the clavicle, without sacrificing the clavicle or without injury to the fibers of the pectoralis major muscle. A modified semi-circular incision is made beginning

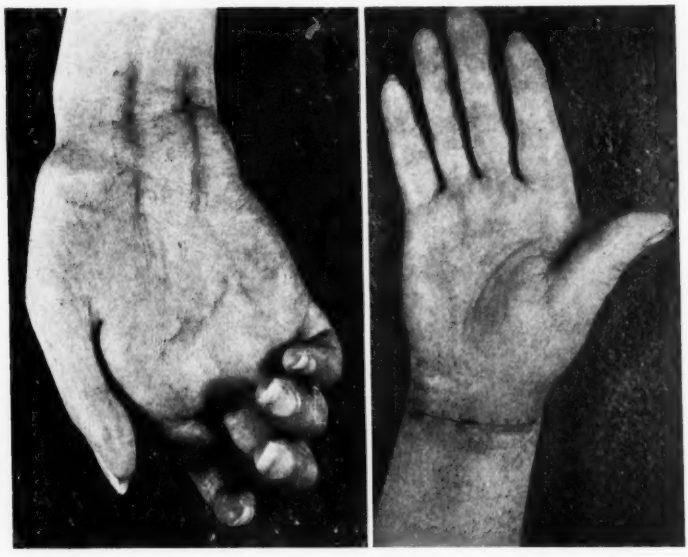

FIG. 14 (*left*). Traction scars resulting from incisions perpendicular to the flexion crease at the wrist.

FIG. 15 (*right*). Incision for exposure of the median and ulnar nerves at the wrist.

above the clavicle near its sternal attachment, and continuing upward and lateralward to the border of the deltoid muscle (part 1, Fig. 13). The incision now curves downwards and crosses the clavicle at its outer third (part 2, Fig. 13); it is now continued downward toward the axilla (part 3, Fig. 13). When the skin flap is retracted the fascial plane between the deltoid and pectoralis major muscle is clearly discernible. The clavicle is divided at this point; the lateral portion is retracted upwards and the medial portion with

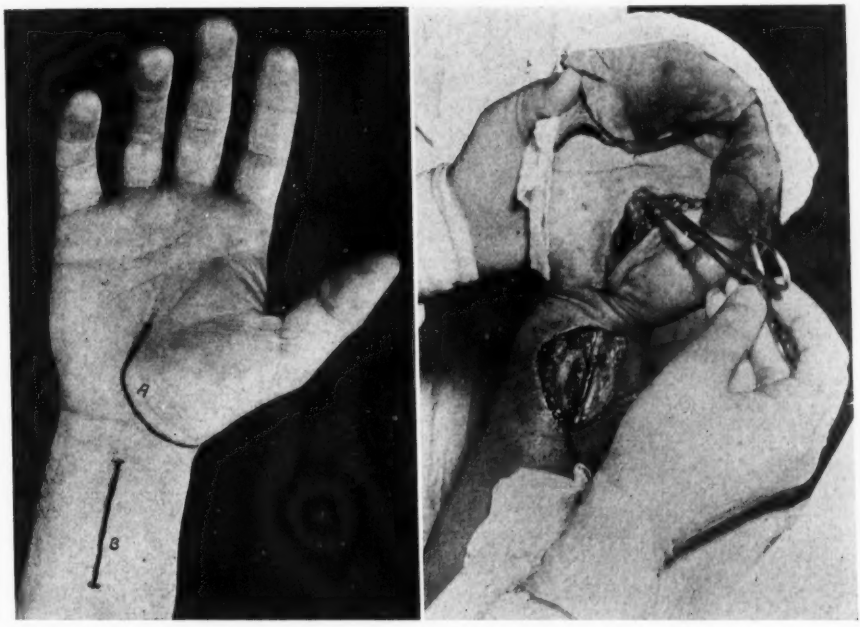

FIG. 16 (*left*). Combined incision for exposing the median nerve in the hand and in the forearm.
FIG. 17 (*right*). Combined incision for exposing median nerve in the forearm and hand. The median nerve, which was severed at the wrist, was rerouted beneath the transverse carpal ligament into the hand.

the pectoralis major attached is retracted downwards and outwards. The pectoral muscle is in this way separated from the deltoid in their normal plane of cleavage, bringing the entire brachial plexus clearly into view. At the termination of the surgical procedure, the clavicle is wired together. This procedure surpasses all other clavicular approaches to the brachial plexus in that the integrity of the shoulder girdle is more easily maintained.

INCISIONS IN THE HAND AND WRIST FOR EXPOSURE OF MEDIAN AND ULNAR NERVES

The most severe and disabling contracture scars are caused by longitudinal incisions crossing the flexion crease and transverse carpal ligament at

the wrist and into the hand (Fig. 14). The median and ulnar nerves on the volar surface of the wrist may be easily exposed by using a transverse incision, in a flexion crease at the wrist as shown in Fig. 15. When using this incision the transverse carpal ligament is also divided in the direction of its fibers. This is an exploratory incision, and should mobilization or transposition be required, the incision is then continued at right angles as far proximally as indicated.[2]

A combined incision for exposing and following the median nerve from the forearm into the hand, consists of two parts as shown in Figs. 16 and 17.

FIG. 18 (*left*). Traction scar at the wrist produced by continuation of forearm incision into the hand.
FIG. 19 (*right*). Incision for exploring the tibial and peroneal nerves in the region of the popliteal fossa.

Part a, Fig. 16 follows a flexion crease in the hand, and is used for exploring the median nerve in the hand. Part b, Fig. 16, a longitudinal incision in the forearm ending at the wrinkle lines in the wrist, is used for mobilization of the nerve in the forearm when necessary. Part b may be continued as far proximally as is found necessary. The small intact area of skin between these two incisions may be easily undermined and the nerve exposed beneath it. A comparable incision is made for exposing the ulnar nerve in the forearm and hand. Adherence to this type of incision would have prevented the contracture scar at the wrist noted in Fig. 18.

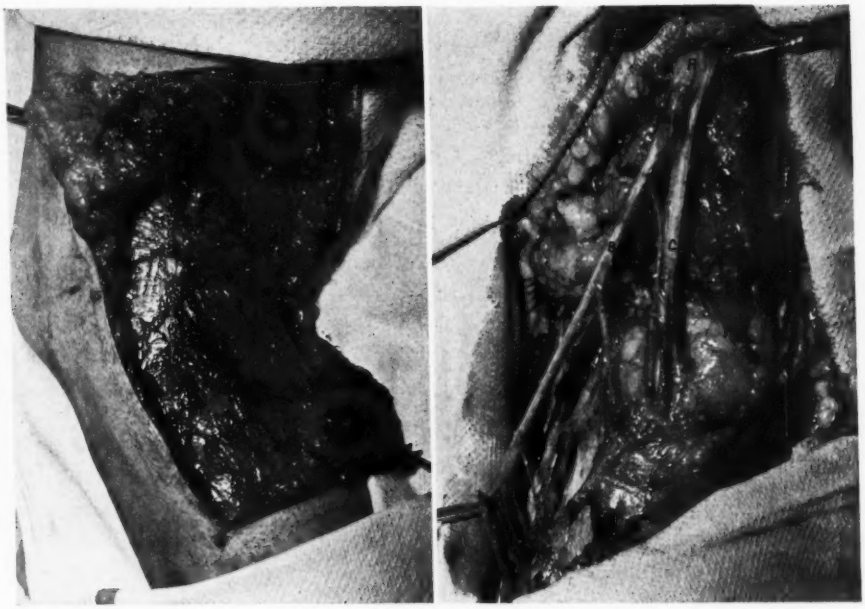

FIG. 20 (*left*). Retraction of skin flaps in the region of the popliteal space to show the operative exposure obtained by use of the Z-shaped incision.

FIG. 21. (*right*). Exposure of the sciatic nerve with its division into the tibial and peroneal in the region of the popliteal fossa. (A) Sciatic nerve. (B) Peroneal nerve. (C) Tibial nerve.

INCISIONS IN THE LOWER EXTREMITY

The Tibial and Peroneal Nerves in the Popliteal Fossa. Here again the incision is divided in three parts as shown in Fig. 19; a longitudinal incision is made along the posterior surface of the thigh, lower third. This incision is carried down to, but not through the flexion crease in the popliteal fossa (part 1, Fig. 19). This incision is used for exposure of the sciatic nerve at the point of division into the tibial and common peroneal nerves. The incision is now carried transversely, at the flexion crease in the popliteal fossa, lateralwards to the border of the head of the

FIG. 22. Traction scar resulting from an incision perpendicular to the flexion crease at the popliteal fossa.

fibula (part 2, Fig. 19); now it is curved downwards lying between the bellies of the gastrocnemius and soleus muscle (part 3, Fig. 19). In this fashion the two flaps are retracted, as shown in Fig. 20, and the tibial nerve may be followed into the calf while the peroneal nerve may be exposed on the lateral surface of the leg at the head of the fibula (Fig. 21). For the exposure of the common peroneal nerve alone, the lower third of the incision alone may be used. To omit the modified Z across the popliteal fossa produces the result pictured in Fig. 22.

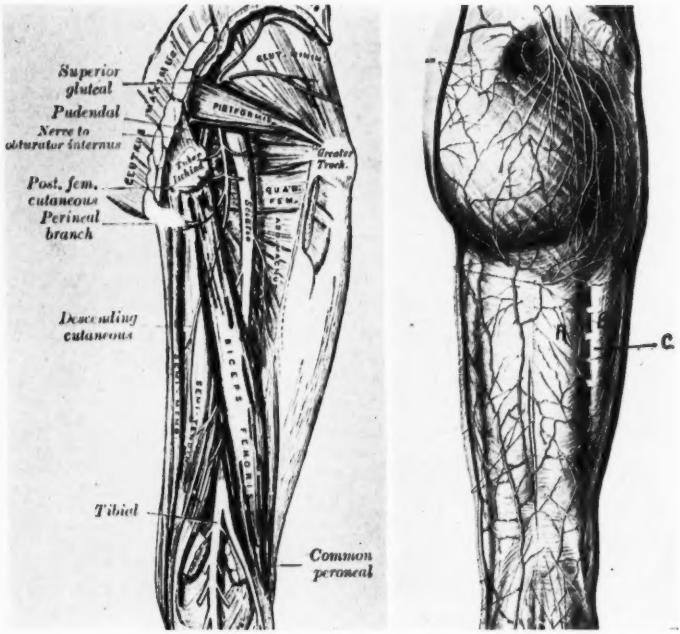

FIG. 23 (*left*). An anatomical drawing to show the relationship of the sciatic nerve to the biceps femoris muscle. (From Gray's *Textbook of anatomy*. Philadelphia: Lea & Febiger, 1930, 22nd ed., p. 958.)

FIG. 24 (*right*). Incision for lateral approach to the sciatic nerve in the thigh, in (C) plane of division between (B) vastus lateralis and (A) biceps femoris muscle. (From Sobotta's *Atlas of human anatomy*. New York: G. E. Stechert & Co., 1932, 3rd ed., p. 608.)

THE LATERAL APPROACH FOR EXPOSING THE SCIATIC NERVE IN THE THIGH

A review of the anatomy of the sciatic nerve in the thigh (Fig. 23) reveals that the massive biceps femoris muscle crosses the sciatic nerve in the middle third of the thigh from mesialward to lateralward. This is easily understood since the muscle originates from the ischial tuberosity, while the insertion is over the head of the fibula and lateral condyle of the tibia as shown in Fig. 23. The sciatic nerve below the level of the gluteal fold lies

beneath the lateral border of the biceps femoris muscle in the upper two-thirds of the posterior thigh, rather than in the midline between the biceps and semitendinosis muscle. When the accepted midline incision is used along the posterior surface of the thigh, the biceps femoris muscle must be separated from both the semimembranosis and semitendinosis muscles, before the nerve can be exposed, and in addition it will often be necessary to sever part of the attachment of the biceps muscle from the ischial tuberosity before the upper portion of the sciatic near the gluteal fold is adequately exposed. The lateral incision, therefore, is a longitudinal incision placed in the plane of division between the vastus lateralis and biceps femoris muscle,

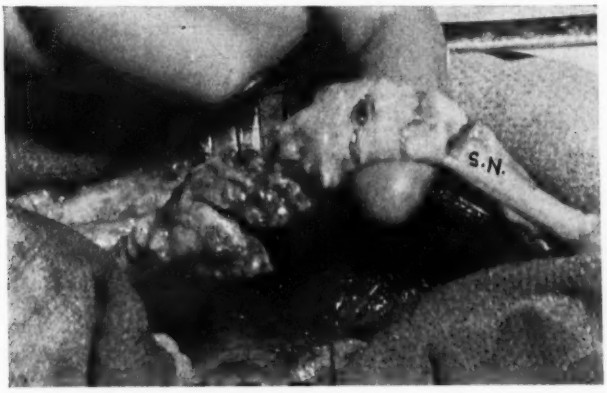

FIG. 25. Exposure of the sciatic nerve in the thigh by the use of the "lateral approach." (S.N.) Sciatic nerve. (B.M.) Biceps femoris muscle.

as shown in Fig. 24. By gently elevating the biceps femoris muscle the sciatic nerve is easily exposed, as shown in Fig. 25.

EXPOSURE OF THE SCIATIC NERVE BENEATH THE GLUTEUS MUSCLES

The "question mark" or sickle-shaped incision of Stookey[8] is used for exploration of the sciatic nerve from its exit at the sciatic notch to the gluteal fold. A curved incision is made beginning in the mid-gluteal region at the level of the sacro-iliac joint and continuing downward and outward over the region of the greater trochanter of the femur. The incision is now curved downward and then inward along the gluteal fold, to the mid-thigh and continued down along the mid-thigh for about 5 cm. as shown in Fig. 26. The gluteus maximus muscle is split along the course of its fibers, all the way to the fascial and tendinous insertion at the iliotibial band over the greater trochanter. Great care is now used to sever the gluteus maximus muscle at its fascial insertion. The entire muscle is now reflected medialwards.

For convenience the Stookey incision is divided into three parts. The first (part I, Fig. 26) may be used alone to expose the sciatic nerve at the

FIG. 26 (*left*). Incision of Stookey for exploration of the sciatic nerve from its exit at the sciatic notch to the gluteal fold.

FIG. 27 (*right*). Incision for exposure of the sciatic nerve at the gluteal fold.

sciatic notch, especially when a neurolysis alone is indicated. The lower third (part III, Fig. 26) may be used alone for sciatic nerve exposure near the gluteal fold. When the lower incision alone is used (Fig. 27), only a small portion of the gluteus muscle insertion is severed.

EXPOSURE OF THE TIBIAL NERVE

The tibial nerve lies beneath the calf muscles, the gastrocnemius and soleus and adjacent to the posterior tibial muscle.[5] It may best be reached by a longitudinal incision over the mesial surface of the leg[3] and approached beneath the fascial plane of the soleus muscle as shown in Fig. 28 (incision of Cliffton). This incision may be continued proximally when it is found necessary to reflect the soleus muscle in the presence of a very extensive tibial nerve defect.

It must *never* be approached through the calf by a posterior incision as shown in recent literature since the muscles are so constructed that they cannot be split in the direction of their fibers. The trauma

FIG. 28. Incision for exposure of the tibial nerve in the leg.

produced by penetrating the calf muscle results in a severe postoperative reaction as well as severe disabling adhesions between the calf muscles, and definitely impedes plantar flexion of the foot. Fig. 29 is a cross section of the leg in its upper 3rd[10] showing the anatomy of the tibial nerve and the new approach.

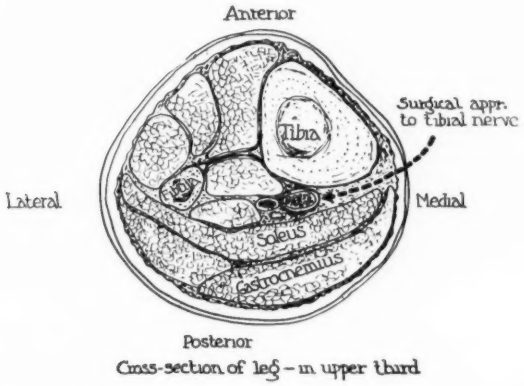

FIG. 29. Cross section of the leg in its upper third showing the relationships of the tibial nerve, and the surgical approach.

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A REPORT ON THE TREATMENT OF CRANIOCEREBRAL WOUNDS IN AN EVACUATION HOSPITAL

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THIS REPORT will deal with the method and the results of treatment in a series of 126 consecutive penetrating wounds of the skull and brain treated in an American evacuation hospital between 1 September 1944 and 15 April 1945. It is recognized that the series is small, but since it represents all of the craniocerebral wounds admitted to one hospital attached to one army operating in France and Germany, it represents a fair cross section of neurosurgical casualties as they were received, without triage, in the most forward medical installation where definitive neurosurgery was done. We feel that it may add something to the overall conception of the treatment of craniocerebral injuries in this war. The total picture will be gained only when much larger statistics with long-time follow-ups are forthcoming from installations in the rear.

TYPE AND SOURCE OF PATIENTS

During the period covered by this report, 1 September 1944 to 15 April 1945, the hospital admitted over 19,500 patients of all types, both medical and surgical. This series of 126 cases includes every pentrating wound of the brain and every penetrating wound of the skull with depressed fracture without dural penetration admitted to the hospital, whether or not surgery was performed. Penetrating wounds of the scalp with or without linear skull fracture and closed type head injuries secondary to automobile accidents, etc., are not included. Table 1 shows the breakdown of patients, both operated and non-operated, into those with and without dural penetration.

TABLE 1

Craniocerebral wounds

	Operated	Non-operated
Compound skull fracture with dural and brain penetration	90	12
Compound-depressed skull fracture without dural penetration	24	0
TOTAL	114	12

American soldiers, German prisoners of war, and civilians make up the series. Most of the military patients were admitted from medical clearing companies, or, as in the case of some with abdominal and thoracic wounds, from a field hospital, which may be operating several miles ahead of the evacuation hospital. Several of the civilians passed through similar channels, but a few were admitted directly to the preoperative ward of the hospital because their wounds were incurred in close proximity to the hospital, secondary to mine explosions. Two of the prisoner patients, unoperated

* Released from military service; now at 1502 Carew Tower, Cincinnati, Ohio.

upon several days after receiving their wounds, were admitted from a captured German hospital. One American was received from another evacuation hospital after debridement of what was thought to be a superficial scalp laceration, and another was a recaptured prisoner who was admitted from a German hospital 11 days after injury.

RESULTS

Of the total admissions, 114 had one or more craniectomies each while in the evacuation hospital, with 14 postoperative deaths or a surgical mortality of 12.3 per cent. Twelve patients died before reaching surgery making a total mortality, based on admissions, of 20.7 per cent. The time interval between the receipt of the wound and the initial surgery is shown in Table 2.

TABLE 2

Interval between injury and surgery

Time of operation	American	German	Civilian
Average	23 hours	56 hours	25 hours
Range	6 hours–12 days	7 hours–6 days	8 hours–2 days

It will be noted that the preoperative interval is practically identical in the American soldier and civilian groups but is more than double in the German prisoner group. This is due to the fact that more of the latter group arrived at the hospital late rather than to any sorting or selection after admission.

The average postoperative hospital stay was 7 days, with a range between 2 and 24 days. It is especially notable that 13, or exactly one-half, of the patients died within 24 hours (Table 3) from the time they received their

TABLE 3

Time of death in operated and non-operated cases

	Total	Operated	Non-operated
24 hours or less	13	3	10
24–48 hours	3	2	1
48–72 hours	6	5	1
1–2 weeks	3	3	0
3 weeks	1	1	0

wound and only 4 survived more than 3 days. Two of the surgical deaths occurred on the operating table. This should be of interest in assessing mortality statistics from neurosurgical centers further in the rear, for it is obvious that a large group of patients, except under the most favorable evacuation system, would not reach a center far in the rear. It is a well recognized fact that, in general, patients with penetrating wounds of the head should be evacuated far enough to assure good, complete, definitive neurosurgery with all of the necessary modern surgical armamentarium available, such as the electrocautery, suction, special instruments and supplies, etc. In the American Army, this has been established at the level of the evacuation hospital.

Sixteen, or 14 per cent, of the surgical patients either were infected upon admission or developed infection before discharge. Unfortunately, culture facilities were not available much of the time and the diagnosis of infection

had to be established on clinical evidence alone. Two were classified as superficial scalp infection, 4 as subgaleal, and the remainder as cerebral abscess, meningitis, or both. One patient was admitted with osteomyelitis and brain abscess, and two others were admitted with frank abscesses. In one case the diagnosis, confirmed by culture, was Klebsiella pneumoniae, type A meningitis and the patient succumbed 21 days postoperatively.

In the operated series, there were 39 cases in which multiple lobes of the brain were involved (Table 4). Nine of the 14 deaths occurred in this group. Of these injuries, 26 were bicerebrate in type and 8 definitely involved the ventricles and 3 others probably did so. Six were traversing or perforating wounds, 12 were tangential or gutter type wounds, and 21 were penetrating.

TABLE 4

Type of injury with breakdown of mortality

	Total			Surgical			Non-surgical		
	No.	Mort.	Per cent	No.	Mort.	Per cent	No.	Mort.	Per cent
Skull fracture with penetration of brain	102	25	24.5	90	13	14.4	12	12	100
Single lobe				51	4				
Multiple lobes				39	9				
Skull fracture without dural penetration	24	1	4.2	24	1	4.2			
TOTAL	126	26	20.7	114	14	12.3	12	12	100

There were 51 cases in which a single lobe was injured; 31 of the wounds were penetrating in type and 20 were tangential. Six involved the ventricle. There were 4 deaths in this group.

The dura was not penetrated in 24 cases, but in 12 of these there were definite neurological abnormalities on examination and there was one death from intracerebral hemorrhage.

In the entire series, 9 cases were complicated by extradural hematoma and 7 by subdural hematoma. Two of the latter were bilateral. In 3 cases the sagittal sinus was lacerated and in 4 the lateral sinus was torn. There were complicating fractures of the mastoid in 2 cases and of the frontal accessory sinuses in 10.

Three patients developed postoperative generalized convulsions, all of which were controlled by the administration of phenobarbital.

No less than 34 patients in the series had complicating injuries of other organs in addition to their cerebral wound. These included simple soft tissue wounds, fractures of the long bones, penetrating wounds of the abdomen and thorax, maxillofacial and eye wounds, and one pregnancy at term.

TREATMENT

Immediately upon admission to the preoperative ward, it is of paramount importance that the general physical and neurological status of each patient be assessed quickly in order that a priority list for surgery may be set up. This is particularly important in times of stress when several patients may be admitted at one time. Whenever possible this initial examination should be done personally by a member of the neurosurgical team. When not possi-

ble, it can be delegated to a properly instructed receiving officer. The examination should include the removal of all bandages in order that all wounds can be evaluated against and with the craniocerebral injury and a brief but thorough neurological examination. The success of the entire program of treatment well may hinge upon this initial assessment, and too much importance cannot be assigned to it. The patients may be separated into three main categories on clinical grounds alone: the critically wounded, the seriously wounded, and the less seriously wounded.

The critically wounded patients are recognized easily. Many arrive in surgical shock, generally due to complicating wounds, either compound fractures, abdominal or thoracic wounds, in addition to their cerebral injury. Others appear moribund upon admission either because of increased intracranial pressure secondary to hemorrhage or because of extensive trauma to vital cerebral centers. The following case reports illustrate the treatment in this group of patients.

CASE REPORTS

Case 1. An American soldier was admitted to the hospital at 1700 hours, on 12 November 1944, 3 hours after receiving multiple shell fragment wounds of the right thigh and of the occipital region of the head. At the battalion aid station, he had received first aid treatment and 2 units of plasma, and his right lower extremity had been immobilized in a Thomas splint.

Initial examination showed a practically moribund patient. The peripheral pulse and blood pressure were unobtainable. The pupils were dilated and did not react to light. There was an extensive lacerating wound on the upper anterolateral surface of the right thigh with exposure of the femur. There was a tangential gutter type wound, measuring 10×6 cm., in the left occipitoparietal region, with cerebral tissue herniating. One unit of plasma was given immediately through a cannula tied into the right antecubital vein followed by a total of 1500 cc. of whole blood and 2000 cc. of Alzever's solution. The systolic pressure was obtained then between 90 and 100, and radiographs were taken which showed a badly comminuted fracture of the neck of the right femur, and a comminuted fracture of the left parietal bone with indriven bone fragments and two metallic foreign bodies within the parietal lobe.

He was taken to the operating room at 0500 hours on 13 November 1944 where debridement of the thigh wound was carried out without anesthesia, and the extremity was securely immobilized in a hip-leg plaster spica. During the operation and upon return to the ward, an additional 1500 cc. of blood and 500 cc. of Alzever's solution were given.

Reexamination at 1400 hours revealed a still comatose but restless patient, but the blood pressure had stabilized at 110/70 and the pulse was 88 and of good quality. The right pupil measured 3 mm. and reacted to light. The left pupil was dilated to 8 mm. and showed no reaction to light. In addition there was a right facial palsy and a paresis of the right arm. The right lower extremity could not be evaluated. There was total areflexia.

At 1500 hours, left occipitoparietal craniectomy was done under ether-oxygen endotracheal anesthesia with debridement of the comminuted fracture and removal of the indriven intracerebral bone fragments. A moderately large subdural and intracerebral hematoma was evacuated. During this operation, 500 cc. of blood in 500 cc. of Alzever's solution were given.

Following surgery the patient's general and neurological condition improved rapidly. On 14 November 1944 he was conscious although slightly irrational and he was taking nourishment by mouth. He was evacuated to a general hospital on 19 November 1944. A personal communication to the authors reveals that secondary craniectomy was required at the general hospital, but that following this and following treatment of the fractured femur, he was evacuated to the United States in good condition.

Comment. There can be no doubt that this patient survived because it was recognized that regardless of the severity of his intracranial wound, it did not account for the surgical shock, and the femur was firmly immobilized in plaster after initial shock treatment before the neurological problem was attacked.

Case 2. An American soldier was admitted to the hospital at 1900 hours, on 31 December 1944, 7 hours after receiving multiple shell fragment wounds of the head, face, thorax, right lower extremity and right forearm. There was a traumatic amputation of the right foot. One small foreign body had penetrated the right lung, but there was no appreciable hemothorax. X-ray of the skull revealed a comminuted fracture of the right temporal bone with indriven bone fragments and a metallic foreign body, 8 mm. in diameter, in the right frontal lobe.

On admission, he was semi-comatose. Blood pressure was 118/80, pulse 68, and respirations 20. The pupils were equal in size and reacted normally. A left hemiparesis was present and the deep reflexes were exaggerated on this side. The left abdominal reflexes were absent, and there was a positive left Babinski sign. Coma deepened after admission, and the pulse rate dropped to 50.

Right-sided intracranial hemorrhage was suspected, and he was taken to the operating room at 0030 hours on 1 January 1945. Craniectomy was performed under ether-oxygen endotracheal anesthesia. No extradural bleeding was encountered, but there was a considerable amount of subdural and intracerebral clotted blood found and evacuated. Several bone fragments were removed, but the foreign body was not accessible and was left in place. Debridement of the other wounds was accomplished, and skin traction was applied to the amputation stump.

Later on the operative day, the patient was conscious and fairly rational. He continued to improve and was discharged to a general hospital 12 days later.

Comment. This patient illustrates one of the few instances when immediate neurosurgical intervention is imperative, that is, when progressive intracranial hemorrhage is suspected. It has long been an established fact, however, that there are few operative neurosurgical emergencies and, quite to the contrary, the seriously injured patient may be benefited by a 12- to 24-hour delay, during which time his intracerebral dynamics may have an opportunity to stabilize.

Case 3. An American soldier was admitted to the hospital at 1200 hours, on 30 October 1944, within 3 hours after receiving a penetrating shell fragment wound of the left parietal region. He was in deep coma upon admission, and respirations were Cheyne-Stokes in character. Blood pressure was 130/60 and the pulse varied between 42 and 88. There was a 1-cm. penetrating wound of the posterior left parietal scalp. The pupils were widely dilated and fixed to light. The knee kicks were elicited, the right being slightly greater than the left. Otherwise the deep and superficial reflexes were absent. There were no plantar responses. X-ray revealed a comminuted fracture of the left parietal bone with several indriven bone fragments and a metallic foreign body within the right frontal lobe. An extensive fracture line extended from the defect anteriorly into the left frontal bone and crossed the midline to the right.

The patient was taken to surgery within a few hours after admission, and under local 1 per cent procaine infiltration anesthesia craniectomy was done. The left cerebral hemisphere was found to be under great tension and several bone chips were expressed with a large amount of blood and macerated cerebral tissue. The missile tract was inspected under direct vision and was found to communicate with the lateral ventricle. The ventricle was filled with fresh blood, but the bleeding point could not be found. A trephine opening was made in the right frontal region, and a large subdural hematoma was evacuated on this side. Ventricular puncture was made, again encountering blood. Through-and-through irrigation of the ventricular

system was accomplished with warm normal saline solution until the returns were clear. The wounds were then closed, and the patient was returned to the ward, where he expired at 2130 hours on the same day.

Necropsy showed a traversing ventricular wound with extensive cerebral damage and midbrain hemorrhages. The ventricular system was filled with clotted blood but the source of hemorrhage could not be determined.

Comment. This case might be classified as desperate surgery, and perhaps a wiser surgeon would not have operated upon the patient. In our series, no patient admitted with Cheyne-Stokes respirations and with widely dilated fixed pupils survived. We have not been conservative in these cases, except in times of stress, for fear that some operable lesion might be overlooked, such as an extradural hemorrhage.

The seriously ill group of patients includes those with severe penetrating wounds of the brain, often with a protruding cerebral fungus and with, or without, associated complicating injuries, who are admitted in fairly good general physical condition, but whose vital signs do not suggest any immediate danger to life. These patients are given a high priority for treatment, only slightly lower than the preceding group. Case 4 illustrates this group.

Case 4. An American soldier was admitted to the hospital at 1725 hours, on 1 February 1945, 5 hours after receiving a perforating wound of the skull and brain. He was conscious at the time of initial examination, but was extremely uncooperative, continually tearing at his bandages and attempting to get out of bed. Blood pressure was 120/40, pulse 116, and respirations 28. His respirations were somewhat stertorous, and there was much mucus in the tracheobronchial tree. The pupils were equal and reacted to light. A divergent strabismus was present and he was unable to focus his eyes. He was completely aphasic on the motor side, but appeared to understand some of what was going on around him. There was no apparent motor weakness and it was impossible to properly evaluate the deep reflexes, although they appeared equal bilaterally. Both plantar responses were withdrawal in character. There were 2 small 1-cm.-sized wounds of entrance in the left frontal area with cerebral tissue exuding from one of them. There was a larger 4-cm. lacerated wound of exit in the right temporal area, just anterior to the ear, with a large cerebral fungus. X-ray revealed an extensive comminuted and depressed fracture involving the left frontal and parietal bones, and the right frontal, parietal and temporal bones. There was a large collection of intracerebral bone fragments.

Craniectomy was done at 0010 hours, 2 February 1945, under endotracheal ether-oxygen anesthesia. A coronal skin incision was used including the wound of exit in the incision. Debridement was carried out of both the entrance and exit wounds, and the intracranial bone fragments were removed under direct vision. The dural defects were repaired with temporal fascia grafts and both of the wounds and the operative incision were closed tightly.

His postoperative course was smooth, and he was discharged to a general hospital on 11 February 1945. At the time of discharge, he was up and about the ward, but a total motor aphasia persisted. A personal communication from the neurosurgeon at the general hospital on 26 March 1945 stated that he had been evacuated to the United States with approximately 20 per cent motor aphasia persisting.

Comment. It is surprising that this man with a perforating wound did not exhibit more neurological abnormality. However, the missile tract was anterior to the motor areas and it is probable that the aphasia was secondary to severe contusion rather than to actual direct destruction of the motor speech center.

The less seriously wounded group of patients includes those with compound depressed skull fractures without dural penetration and those with less extensive penetrating wounds of the brain who show little or no neurological or general physical abnormality. These patients are conscious and may even be ambulatory, as illustrated by Case 5. They can be given a low priority for surgery in times of stress and may be evacuated further to the rear at such times.

Case 5. An American soldier was admitted to the hospital at 1600 hours, on 14 April 1945, approximately 16 hours after receiving a penetrating shell fragment wound of the head. He was admitted as a walking patient and examination revealed a small 1-cm. wound of entrance in the left posterior parietal region. General physical and neurological examinations were normal. X-ray of the skull showed a comminuted depressed fracture of the left parietal bone with a metallic foreign body within the left parietal lobe.

At 1800 hours on the same day a craniectomy was done through a left parietotemporal skin flap under local 1 per cent procaine anesthesia. The site of fracture, lacerated dura and brain were debrided, and several small indriven bone fragments were removed from the parietal lobe. The missile was not removed. The dura was closed using a sliding flap of periosteum, and both the wound of entrance and the scalp incision were closed tightly.

Comment. This patient's course was uneventful and he continued to be ambulatory after the first postoperative day.

DISCUSSION

Good X-rays of the skull are indispensable to the performance of adequate debridement of the fracture and removal of indriven bone from the brain, for it is essential that the surgeon know the exact position and extent of all metallic and bony fragments. Stereoscopic views taken in both the anteroposterior and lateral views are desirable but too often are not practicable in the acutely ill, extremely restless, uncooperative patient. In cases involving the frontal sinuses and orbital walls, posteroanterior views are required in addition.

In preparation for surgery, all patients are typed and crossmatched with 1000 cc. of blood. When whole, undiluted, blood is available, an intravenous drip of 5 per cent dextrose in normal saline is started at the beginning of the operation and is supplemented by the blood if necessary during the course of the operation. When blood, diluted with Alzever's solution, is used, the blood mixture is started directly in order to keep the total fluid intake at a lower level.

We have found that the total preparation of the head is best done in the operating room since it is difficult to maintain the necessary aseptic precautions in the face of an open wound in the preoperative tent.

Ether-oxygen endotracheal anesthesia has been used almost routinely in the restless and uncooperative patients, and 1/100 gr. of atropine sulphate is used as the only preoperative medication. In the cooperative, non-apprehensive patient local infiltration and block of the scalp with 1 per cent procaine has been used. In these patients, nembutal or seconal, gr. iii, is given 2 hours preoperatively and morphine sulphate, gr. $\frac{1}{6}$, 1 hour preoperatively without ill effect.

Tripod incisions, which were used extensively by neurosurgeons in the last war, have been discarded almost universally by neurosurgeons operating in the European Theater of Operations. Occasionally, the nature of the wound is such that three-cornered skin junctions are unavoidable. Too often these become the site of annoying cerebrospinal fluid fistulas. Before positioning and draping the head, preparatory to surgery, it is essential to carefully plan the incision so that final tensionless closure may be effected at the site of the cranial defect. Either linear incisions including the wound or curved incisions around the wound have been found most desirable by the majority of surgeons operating in this theater. The latter allow wide exposure of the cranial defect and at the time of closure may be rotated in cases of large skin defects in order to accomplish closure. This may leave a scalp defect elsewhere but this will be over intact pericranium and skull, and skin grafting may be accomplished either immediately or at a later date. Relaxing incisions after extensive undermining of the scalp have been recommended by some, and we have used them a few times. Reports reaching us from installations in the rear area lead us to believe that this is much less satisfactory than the use of rotated skin flaps. In all frontal cases involving the frontal sinuses, a coronal incision just anterior to the ears has been used. This provides excellent exposure of the pathology and gives the best cosmetic result.

Debridement of the wound and of the comminuted fracture follows good surgical principles. All dirt, hair, and debris are removed together with badly comminuted bone fragments. The cranial defect is enlarged enough to expose normal-appearing dura mater. Troublesome bleeding from dural and cortical vessels is controlled either by the judicious use of the electrocoagulating current or by the application of silver brain clips. Oozing from the brain substance and from torn dural sinuses can be controlled readily by the application of fibrin foam. Clotted blood, dirt, hair and all indriven bone fragments must be removed from the brain together with all macerated devitalized cerebral tissue. At the stage when these patients are operated upon in the forward hospital, it is sometimes difficult to determine the exact direction of the missile tract because of existing cerebral edema. The gentle introduction of a soft rubber catheter will aid in initial orientation and avoid unnecessary trauma. Once the tract is determined it should be explored under direct vision until all bone fragments have been found and removed. This is accomplished with the aid of gentle suction. Once a core of necrotic brain and blood clot has been removed there is generally a tract present which will admit the gloved finger. Although any direct palpation of the cerebrum has been condemned, it is believed that the gentle introduction of the finger along the tract often will detect one or more good-sized bone fragments in the wall which would otherwise go undetected and would become a potential source of latent infection and brain abscess formation with resultant greater cerebral damage than will result by this maneuver. This is mentioned with some temerity and with the caution that it must not be used as a general

method of blind palpation of the brain, but only as a supplemental procedure in certain cases after a well defined cavity has been outlined under direct vision. Immediate radiologic check at the operating table may be a better answer to this perplexing problem of retained bone fragments, but we have been unable to do this in our hospital.

Chemotherapy has made tight closure of the dura and scalp without drainage possible in almost all penetrating wounds of the brain in this war. We believe that this should be accomplished in all cases where operation is performed within 72 hours after injury and may be done even in later cases when frank infection is not present. In old cases, with obvious infection and without ventricular penetration, the dura is best left open and the scalp closed. All cases with ventricular communication should have water-tight dural closure. Dural defects are best repaired with sliding or rotated periosteal flap grafts, thus preserving the blood supply of the graft. When this is impossible free grafts of periosteum, temporal fascia, or fascia lata are preferred in that order. The scalp should be approximated carefully without tension, using buried galeal sutures of fine silk and interrupted cutaneous sutures of the same material.

We have not used local sulfonamide drugs. In some cases 20,000–30,000 units of penicillin have been introduced into the intracerebral operative cavity after closure of the dura. All patients have received 20,000 units of penicillin intramuscularly throughout their postoperative course, plus sulfadiazine for at least 72 hours or until they have been fever free for 24 hours. Some have received penicillin intrathecally in 10,000 unit doses on one or more occasions.

While receiving sulfadiazine, the fluid intake has been maintained at 3000 or more cc. daily, and the blood and urine have been examined each second day. Otherwise the fluid intake has been maintained at approximately 2000 cc. daily. Dehydrating agents have not been used. Opiates have not been used postoperatively. Sedation has been restricted to barbiturates, notably phenobarbital, sodium amytal, and paraldehyde.

On return to the ward from surgery, the patients are placed in semi-Fowler's position and are turned every two to three hours as long as they are comatose or unable to turn themselves. If possible, they are gotten out of bed into a chair on the second or third day.

SUMMARY

A series of 126 penetrating wounds of the skull and brain, treated in an army evacuation hospital in Europe, has been reported. Of these, 114 were operated upon with a surgical mortality of 12.3 per cent. The cases were classified as to type, and the treatment was discussed.

WATER CONTENT OF THE BRAIN AFTER CONCUSSION AND ITS NONCONTRIBUTORY RELATION TO THE HISTOPATHOLOGY OF CONCUSSION*

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STUDIES of concussion of the brain and spinal cord in experimental animals have demonstrated structural alterations in nerve cells of a permanent nature and significant magnitude. In guinea pigs and cats it was found that some of the larger neurons of the brain stem[12] as well as the spinal cord[3] underwent subtle structural alterations immediately after striking an adequate blow. Within 24 hours these same large neurons as well as many smaller cells began to undergo degenerative changes. Primary motor and sensory neurons were relatively unaffected. The greatest structural change appeared in interneurons of the reticular formation, vestibular nuclei and red nuclei. The chromatolysis resulting from concussion was shown to be different from that which followed asphyxiation and that which occurred as a result of axon section. Concussion was differentiated from contusion and skull fracture. Hemorrhages and vascular changes were not observed after simple concussion of the brain or spinal cord. After a series of concussive blows it was shown that permanent cell loss in certain nuclei of the brain stem amounted to more than 50 per cent.[11]

Our experimental investigations of brain and spinal cord concussion suggested that the severe and irreversible cell changes were due to primary injury at the time of concussion. This theory was supported by the observation of immediate† cytoplasmic disorganization during concussion and was strengthened by failure to observe afterwards any histological evidence of edema or vascular alterations that might have been primary to the cytopathology characterizing postconcussion. However, it was reported by some investigators that swelling of the brain of experimental animals occurred after concussion[10] and that an increase in cerebrospinal fluid pressure was demonstrable after head trauma.[6,7,10] Others, notably Ferraro,[2] claimed to have found numerous cytopathological changes following edema produced experimentally. Thus, it became evident that we should have to test the possibility that the cytological changes and loss of cells that we have re-

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† It should be noted that in a recent review[1] of the subject of cerebral concussion the statement that Windle, Groat and Fox[12] "failed to find any immediate histological change" is in error.

ported might have been brought about by an increase in pressure upon the brain due to swelling or edema rather than to a primary injury of the nerve cells themselves. That is the concern of the present report.

Guinea pigs were used for this study because methods of producing concussion had been perfected and histopathological changes in the brain had been most carefully worked out in this species. The first task was to devise an accurate method for detecting small changes in water content of the brain which might be encountered after concussion.

The brain was placed in a previously tared beaker and weighed. It was immediately covered with 25 cc. of acetone under which it was subdivided into pieces of approximately 2 c.mm. volume to facilitate removal of the water. The material was allowed to stand in acetone for 60 minutes. The fluid was then poured into another tared beaker and a second 25 c.c. portion of acetone was poured over the tissue. After 45 minutes this was decanted into the beaker containing the first 25 cc. portion. A third extraction with 25 cc. of acetone was

TABLE 1

Comparison of water content of brain halves

Animal*	Wt. of Half Brain (gm.)	Water Content of Half Brain (per cent)
A	1.8064	78.66
A′	1.9475	78.67
B	1.8101	78.69
B′	1.8355	78.66
C	1.8400	78.70
C′	1.8243	78.67

* Halves of three brains are indicated by A and A′, B and B′, C and C′.

performed, this time for 30 minutes, and the supernatant fluid was added to the first two portions.

The beaker containing the acetone washings was placed in an oven at 70°C. and the beaker containing the dehydrated, acetone insoluble brain residue (including phospholipids) was placed in a vacuum oven at 60°C. As soon as the acetone had evaporated (about 2½ hours) from the beaker containing the water, fats, cholesterol and its esters and galactolipids, it also was placed in the vacuum oven. Frequent weighings determined that the substance in the beakers reached constant weight within 24 to 28 hours. The low temperature, low air pressure and short time in the oven were used to minimize oxidation of phospholipids. It was found that the dried residues increased slightly in weight if allowed to stay in the oven longer than 36 hours.

Methods of killing the guinea pig and removing its brain were standardized. The untreated animal was decapitated on a guillotine. The skin and muscles of the head were quickly removed and an incision was made through the atlanto-occipital membrane severing the spinal cord about 1.5 to 2 mm. caudal to the obex of the medulla oblongata. The bone was promptly removed over the cerebellum and then over the cerebrum. The dura was laid back and the brain quickly scooped intact from the cranium with a flat, blunt instrument. It was placed immediately in the beaker provided for it. The total time elapsing between decapitation and weighing the specimen was 1½ to 2 minutes.

Preliminary experiments were performed to ascertain the accuracy and uniformity of this method for quantitative determination of water in brain

tissue. In the first experiment 3 untreated guinea pigs were used. Brains were removed and divided into halves as evenly as possible by a midsagittal section. Results are shown in Table 1. The experiment demonstrated that the percentage of water in two halves of the brain ascertained separately by this method are very much the same. The maximum difference was 0.03 per cent, which means that the maximum error in a determination will be ± 0.03 per cent.

In a second experiment 3 additional brains were halved. The water content of each half was determined after adding one drop of water to one-half of each brain. The results are shown in Table 2. The calculated percentage

TABLE 2

Recovery of water added to one brain half in each of three guinea pigs

Animal*	Wt. of Half Brain (gm.)	Wt. of Half Brain plus Added Water (gm.)	Water in Control Half (per cent)	Calculated Water in Watered Half (per cent)	Actual Water in Watered Half (per cent)
D	1.8569		78.39		
D′	1.8884	1.9247		78.80	78.82
E	1.8635		78.64		
E′	1.8912	1.9273		79.04	79.07
F	1.8239		78.45		
F′	1.8137	1.8504		78.87	78.87

* Halves of three brains are indicated by D and D′, E and E′, F and F′.

of water in the watered half was obtained by multiplying the weight of that half before water was added by the percentage of water determined experimentally in the other, control half; adding to this figure the weight of water introduced to the watered half; dividing the sum by the complete weight of the watered half (including the weight of added water); and multiplying by 100. The actual percentage of water in the watered half was determined experimentally by the method already described. It can be seen from Table 2 that the recovery of added water was complete; the greatest variation between water content of control half and watered half was 0.03 per cent, a figure identical with the maximum experimental variation between halves of the same brain established in the preceding test experiment.

The second step in the present investigation was to determine whether the water content of the brain increased after concussion. Young adult guinea pigs were used for this part of the study. Pools of animals comparable in weight, age and sex were selected. Half the animals of each were subjected to concussion and the other half kept for controls. The method used to produce concussion has been described.[12]

Water content of the brain was determined 4, 8, 17, 24 and 48 hours after concussion. Results of the significant experiments at 8 and 17 hours are pre-

sented in Tables 3 and 4. Four hours after concussion there was no increase in water content of the brain. On the contrary, brains of the animals that had received concussions appeared to contain slightly less water than those of the control animals. The mean increase in water content of the brain after

TABLE 3

Water content of brain of guinea pigs 8 hours after concussion

Sex	Animal Pair	Experimental (per cent)	Control (per cent)
Male	49	79.87	78.31
	50	79.21	78.78
	51	79.71	78.73
	52	79.01	79.50
	69	80.34	79.43
	70	80.32	79.53
	71	79.99	79.52
	Average	79.78	79.17
Female	96	78.79	78.37
	98	78.77	78.72
	99	78.99	78.81
	53	78.82	78.90
	72	79.91	78.96
	Average	79.08	78.75

TABLE 4

Water content of brain of male guinea pigs 17 hours after concussion

Animal Pair	Experimental (per cent)	Control (per cent)
134	80.83	78.95
135	79.10	79.19
136	79.27	79.18
137	79.46	78.73
138	78.97	78.82
139	78.82	78.54
140	79.18	79.09
141	78.95	78.02
Average	79.32	78.81

That the mean difference between experimental and control groups at 8 and 17 hours is statistically significant is indicated by an F-test[9] value of 7.31 which, for 1 and 37 degrees of freedom, is significant at almost the 1 per cent level.

8 hours was a little less in the group of females than in the group of males but was statistically significant in both (Table 3). At 17 hours a somewhat similar elevation in water content was observed (Table 4). The maximum mean increase after concussion amounted to only 0.5 per cent of wet brain weight or 19 mg. of water—approximately half a drop. At 24 and 48 hours no sig-

nificant differences in water content of the brains of animals receiving concussions and their controls were determined.

Although water content of the brain appeared to increase only slightly after concussion, the increase being so little that it seems scarcely possible to account for the marked cytopathology on this basis, it was thought that an increase in the cerebrospinal fluid pressure outside of the brain substance might have occurred and that this might have been severe enough to produce cytological changes. In order to check this proposition another pool of animals was selected. Half of them were given concussions and kept with their controls for a period of 15 hours. Each animal was then anesthetized with nembutal, placed in a prone position with the head ventroflexed, and the dorsal atlanto-occipital membrane was exposed. Through this a number 23 gage hypodermic needle was inserted into the cisterna magna. The external opening of the hypodermic needle was kept on a level with the cisterna magna. In order to judge any increase in cerebrospinal fluid pressure in the experimental over the control animals the rate at which cerebrospinal fluid appeared in the opening of the needle was observed.

The fluid appeared just as rapidly in the control as in the experimental animals. Furthermore, the animals were left for a period of approximately 2 hours with the needle in position and the amount of outflow of fluid was no different in the two groups. It would appear that no significant increase in cerebrospinal fluid pressure or formation occurred in the type of simple concussion with which we have been dealing. This observation is in close agreement with that of Paterson,[5] who found pressures of 180 mm. of water or less in 70 per cent of his 179 human patients in whom closed head injuries had occurred within 24 hours of the examination. Only 6 per cent of this group of patients showed pressures exceeding 250 mm. of water.

The next step in our study was to produce an increase in water content of the brain equal to or greater than that which was encountered after concussion. If it proved possible to produce brain swelling by nontraumatic methods it would then be possible subsequently to study histologically the brains of animals so prepared and determine whether edema had been responsible for the cytopathology following concussion.

After a number of preliminary experiments the procedure that was selected was that of waterlogging the animals.[8] A group of 20 young adult male guinea pigs weighing approximately 340 gm. was selected. Half of these animals were waterlogged and the other half served as controls. Water in the amount of 10 per cent of the body weight of the animal was given by stomach tube every 2 hours for a period of 12 hours. At the end of 12 hours each animal was decapitated and the brain was analyzed for water content by the method previously described.

Results are shown in Table 5. A mean increase in water content of the brains of the waterlogged animals amounted to 0.72 per cent, a value higher than that obtained in the concussion experiments. From a statistical standpoint this increase in water of the brain appears to be highly significant.

The final step in the present study was to produce edema by waterlogging guinea pigs for a period of 12 hours and then allowing them to live for a period of time equal to that after which the maximum postconcussional cytological changes were encountered. Four experimental animals and 2 controls were used. They were killed under nembutal 6 days after they had been waterlogged.

The histological procedures were the same ones used in previous studies of the cytopathology of concussion.[11,12] The vascular system was perfused first with a solution of gum acacia in isotonic sodium chloride to wash out the blood, and after this with a solution of

TABLE 5

Water content of the brains of male guinea pigs 12 hours after waterlogging for 12 hours

Animal Pair	Experimental (per cent)	Control (per cent)
1	79.11	78.15
2	78.13	78.07
3	79.19	77.85
4	78.40	78.37
5	78.36	78.17
6	78.97	77.34
7	78.75	77.53
8	79.15	78.61
9	78.43	77.85
10	78.97	78.38
Average	78.75	78.03

The difference between control and experimental groups is extremely significant as indicated by an F-test[9] value of 16.93 which, for degrees of freedom 1 and 18, is about twice as great as necessary at the 1 per cent level.

gum acacia, formalin and sodium chloride to fix the tissue *in situ*.[4] All perfusions were carried out at an hydrostatic pressure comparable with the mean systolic blood pressure of the normal guinea pig (70 mm. Hg).

The brain was removed after fixation and placed in a jar of the fixing solution. Within a few days it was embedded in low viscosity nitrocellulose and sectioned serially at 40μ. Every 10th section from the olfactory bulbs down to the cervical spinal cord was selected for staining. These sections were combined with every 10th section of the brain of one of the control animals. By staining a series of sections of the control animals along with the sections of the experimental animals it was possible to compare the two for differences in stainability which might have resulted from the experimental procedures. The buffered thionin technique[13] was used for staining at pH 4.6.

Critical gross comparison of the sections from the experimental animals with those from their simultaneously stained controls revealed no differences in the appearance of the two brains. A careful microscopical survey was made of each section throughout the series. No vascular alterations were encountered; no hemorrhages were seen. No neurons appeared to be chromatolyzed in any cell group of the brain. The distribution and number of neuroglia ap-

peared to be unaffected. Although no special stains for axis cylinders and myelin sheaths were employed it was quite evident in the preparations stained with thionin that the major fiber tracts of the brain of the experimental animals had not been disturbed. Special search was made throughout the brainstem nuclei which had shown marked cytological alteration 6 days after concussion. No cell changes whatsoever were encountered in the red nuclei, the vestibular nuclei or the cells of the reticular formation of the medulla oblongata. The parts of the brain that are said to be particularly susceptible to anoxia, such as layers III and V of the cerebrum and the Purkinje cell layer of the cerebellum, showed no visible structural changes. Ammon's horn was entirely unaffected. Without knowledge of the identity of the experimental and control sections of the brain, other investigators in the laboratory were entirely unable to distinguish correctly between them.

Thus, it was demonstrated that the administration to guinea pigs of excess water to the point where significant increase in water content of the brain could be determined—an increase in water content exceeding that occurring after concussion of the brain—failed to bring about the cytological alterations that are a strikingly characteristic feature of postconcussion.

SUMMARY

Experiments were performed in guinea pigs to determine whether previously described structural changes and loss of nerve cells in the brain after concussion might be secondary to pressure occasioned by an increase in water content of the brain. An accurate method for quantitative determination of water in brain tissue was developed. Eight and 17 hours after concussion slight though significant increases in the water content of the brains were observed. The maximum increase amounted to only 19 mg. It was also shown that concussion was not followed by an increase in the rate of formation or the pressure of the cerebrospinal fluid.

In other experiments a highly significant increase in water content of the brain was brought about by administering excessive quantities of water by stomach tube. A group of animals so treated was permitted to live for a period of time equal to that after which the greatest postconcussional cytological changes were encountered. In controlled histological studies it was demonstrated that no alterations had been produced. It may be concluded that brain edema after simple concussion in the guinea pig is slight and does not produce the degenerative changes in neurons that are characteristic of postconcussion.

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TREATMENT OF CAUSALGIA

AN ANALYSIS OF 100 CASES

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MANY reports in the literature testify to the efficacy of paralyzing the sympathetic nervous system in the treatment of causalgia, but an adequate body of clinical material is seldom available upon which a comparison can be made of the results of the various methods of producing this sympathetic denervation. A large number of patients with wounds of the extremities have been concentrated in the region in which this hospital has been situated and, since we have been able to follow the great majority of the cases to completion, we have had an unusual opportunity to make such a study. In this series of 100 cases of causalgia of varying degrees of severity we wish to present the results of treatment by means of paravertebral injection of procaine, and preganglionic and postganglionic surgical sympathectomy.

Material. All the patients were Chinese soldiers who had been wounded in the North and Central Burma campaigns. In some instances the treatment of the patient had been completed prior to admission to this hospital, in others the treatment had been started in other medical installations and was completed here, and in the remainder all of the treatment was administered here. The wounds were produced by bullets, grenade or shell fragments in 99 patients while the remaining patient sustained a bayonet wound. The ages varied from 18 to 41 years. The upper extremity was involved in 82 and the lower in 18 patients.

Types of Lesions. The major nerves of the extremity were involved in 81 of the 100 cases and in 10 there was injury only to bones or joints. There was injury to soft tissues only in 5 patients and injury to cutaneous or digital nerves in 4. The types of lesions are shown in detail in Tables 1 and 2. Of the 82 patients in whom the arm was involved there was an associated fracture of one or more of the bones in 35. In the lower extremity, fracture without nerve injury was present in 6 patients while in the remaining 12 there were no instances of associated bone injury.

Time of Onset of the Pain. The characteristic pain appeared within 6 hours after the injury in 48 cases and within 1 to 3 days in another 23 (Table 3). In a few cases its appearance was delayed up to 8 weeks.

Characteristics of the Pain. The spontaneous complaint of the patient was usually "numb pain." Careful questioning through an interpreter was necessary to elucidate the characteristics of the pain in greater detail. Although the limited vocabulary of some of the patients tended to obscure the picture

at times, as a rule a fairly good description of the pain could be obtained. The pain was present constantly in 91 of the cases and was intermittent in the remaining 9. Fifty-five of the patients described the pain as hot or burning in character while the remaining 45 did not associate any sensation of

TABLE 1

Types of lesions producing causalgia of the upper extremity

Median nerve injury in the arm or forearm	17 cases
Multiple injuries of the great nerves	17
Laceration of the brachial artery plus injury to one or more of the great nerves	11
Brachial plexus injury	10
Ulnar nerve injury	9
Radial nerve injury	6
Injury to soft tissues only of arm, forearm or hand	4
Fracture of bone or injury to joint	4
Injury to cutaneous nerves of forearm	2
Injury to digital nerves of hand	2
	82 cases

TABLE 2

Types of lesions producing causalgia of the lower extremity

Sciatic nerve injury	8 cases
Tibial nerve injury	2
Peroneal nerve injury	1
Fracture of bone or injury to joint	6
Injury to soft tissues of the calf	1
	18 cases

TABLE 3

Time of onset of pain after injury

Immediately	36 cases
½ to 6 hours	12
1 to 3 days	23
6 to 14 days	16
3 to 8 weeks	11
Not stated	2
	100 cases

temperature with the pain. Since the rest of the clinical picture of these 45 patients was so nearly identical with the others it seemed obvious that the same fundamental pathologic process was involved. Therefore they were included in the series although in the strict etymological sense the term "causalgia" does not apply to them. The term "minor causalgia" has been used by Homans for cases similar to some of these.

Forty-seven patients described the pain as throbbing or jumping. Twenty-six said it was a sharp pain and 7 called it a tight or swollen pain. The usual description was "constant, throbbing, burning pain." In over three-fourths of the cases the pain was more severe on hot sunny days and less severe on cool rainy days. Cold wet applications had been tried by 44 of the patients on their own initiative with partial relief in 35 and no relief in 9.

Various tests aimed at changing the tone in the blood-vessel walls were employed without giving sufficiently consistent results to warrant further consideration. These tests included raising and lowering the limb, compression of the extremity with a blood-pressure cuff to just above the diastolic and systolic levels, and the injection of adrenalin, prostigmine and nitroglycerine.

TABLE 4

Classification of the patients according to the severity of the pain

Grade of Severity of Pain	Arm	Leg	Total
Grade 1	13	6	19
Grade 2	29	6	35
Grade 3	34	4	38
Grade 4	6	2	8
	82	18	100

Severity of the Pain. This varied from relatively slight pain to extremely severe and incapacitating pain. The patients were all graded on a basis of 1 to 4 (Table 4) according to the following criteria:

Grade 4. These were the patients with the most severe pain. They spent most of the day huddled on their beds protecting the involved extremity. The hand or foot was frequently wrapped in a wet cloth and only the most superficial examination was allowed. The facial expression and bodily attitude was so characteristic that the diagnosis could usually be made with a passing glance. Eight of the patients were included in this group.

Grade 3. These patients likewise complained of severe pain and refused to use the part under any provocation but allowed closer examination of the painful extremity and were somewhat more active about the ward. There were 38 patients in this group.

Grade 2. These patients complained of moderately severe pain, used the extremity a little on occasion and allowed a fairly complete examination to be made. There were 35 patients in this group.

Grade 1. The 19 patients in this group complained of moderately severe pain but used the involved extremity somewhat more actively although still much less than their wounds would otherwise have permitted.

Despite the wide variation in severity, the qualitative characteristics of the pain were so similar that there was little doubt that we were dealing with varying degrees of severity of the same fundamental pathologic disturbance.

Location of the Pain. In 30 of the 43 patients with isolated injury to one of the great nerves of the extremity, the pain was limited to part or all of the skin distribution of the involved nerve; it involved the entire hand or foot in 7 patients and in the remaining 6 the pain extended up the extremity as well. In these last 13 patients the pain was most severe in the palm or sole even though the more proximal portions of the extremity were also involved. In 40 of the 43 cases the nerve was in anatomical continuity even though in some cases the lesion was clinically complete.

In half the patients with injury to various combinations of the great nerves of the arm the pain was limited to the skin distribution of one of the nerves. Less frequently the pain involved the total skin distribution of the involved nerves, and still less frequently the pain was present throughout the entire arm. In 6 out of the 7 cases of multiple nerve injuries in which one of the nerves was completely severed the pain was present only in the distribution of the partially damaged nerve or nerves.

With brachial plexus lesions the pain involved either the palm, finger tips, entire hand or arm rather than the distribution of any one nerve. The addition of injury to the brachial or axillary artery to the nerve lesion did not change either the character or the distribution of the pain. In no instance were the great vessels alone injured.

The location of the pain was not altered by the addition of fracture of one or more of the bones of the arm to the nerve injury. In all 10 of the cases due to bone or joint injury alone, the pain was localized to the site of the wound.

Physical Examination of the Extremity. De Takats has described causalgia as a state of painful vasodilatation of an extremity. In the majority of the cases seen early (within 2 or 3 months after injury) this was strikingly borne out in this series. In some of the patients this vasodilatation persisted for as long as 6 months. Usually after several months the picture changed to one of moderate vasoconstriction in association with various trophic changes of the skin, nails and joints. In the stage of vasodilatation the hand or foot was pink and warm and the skin velvety to the touch. Frequently the vasodilatation was so extreme that tiny drops of blood would appear after gentle testing of sensation with a pin. Perspiration was sometimes decreased and in other cases extremely profuse. This would frequently vary from one examination to the next on succeeding days. Stroking or squeezing the involved area or moving the joints nearly always aggravated the pain. Patients whose pain was severe enough to prevent use of the hand and whose pain was not promptly relieved, quickly developed trophic changes, such as glossy cyanotic appearance of the skin, tapered fingers and periarticular fibrosis. Evidence of these changes was seen as early as 4 weeks after the injury.

Diagnostic Aspect of Paravertebral Injection of Procaine. At least 1 injection was done in 91 of the patients. In cases with involvement of the upper extremity the procaine was injected along the sides of the bodies of the 1st and 2nd thoracic vertebrae. For causalgia of the leg the procaine was placed

along the sides of the bodies of the 2nd, 3rd and 4th lumbar vertebrae. Injections that produced satisfactory sympathetic paralysis of the extremity resulted in complete temporary relief of pain in 73 patients, marked but not complete relief of pain in 16 patients and no relief in 2.

THERAPY

The majority of the patients were treated initially by one or more paravertebral injections of procaine and those not responding adequately were then subjected to surgical sympathectomy. Occasionally the sympathectomy was done without the preliminary paravertebral injections in cases in which the operation was indicated to improve the blood supply to the extremity as well as for the relief of pain.

Two patients (with pain grade 1 and 2) had gradual recession of their pain without any treatment, but both continued to have slight pain during the 3 months they were followed. Neurolyses alone were done 3 and 4½ months after injury in 2 patients (with pain grade 2 and 3) and were followed by gradual lessening of the pain. However there was likewise some pain still present at the time of these patients' discharge 6 months after injury. In another 5 patients (3 with pain grade 2 and 2 with pain grade 3) neurolysis or neurorrhaphy within 2 months after injury produced slight or no relief of pain whereas a subsequent attack on the sympathetic nervous system cured the causalgia. In an additional 30 cases various operative procedures were carried out upon the nerves, after treatment directed at the sympathetic nervous system was begun, without altering the course of the pain.

Paravertebral Injection of Procaine. The injections for the arm were done either according to the technique of White and Smithwick,[1] placing 15 cc. of 2 per cent procaine along the sides of the bodies of the 1st and 2nd thoracic vertebrae or, in the case of most of those done prior to admission to this hospital, by injecting the stellate ganglion through an anterior approach. In the case of the leg, the procaine was placed along the sides of the 2nd, 3rd and 4th lumbar vertebrae. The therapeutic results will be presented separately for the milder cases (grades 1 and 2) and for the more severe cases (grades 3 and 4).

Grades 1 and 2. The 48 patients in this group were treated with 1 to 4 injections each, given 10 days to 5 months after injury. Five patients were completely and permanently relieved after 1 injection. In 7 patients the pain completely disappeared within 4 weeks after the injections. There was moderate relief of pain in 23 patients and slight or no relief in 13. Further treatment was not considered necessary for 26 of these last 36 patients. In 3 of the remaining 10 patients who had little or no lasting relief after the injections (despite complete relief for the duration of the procaine effect) there was moderate relief of pain after subsequent neurolysis. Sympathectomy was resorted to in the remaining 7 patients when it was evident that the relief produced by the injections was inadequate. The duration of the pain

at the time the injections were performed (up to 5 months in this group) did not have any definite relationship to their efficacy in curing the causalgia.

Grades 3 and 4. There were 43 patients in this group and they received 1 to 10 injections over a period from 2 weeks to 5 months after injury. One patient was completely relieved after 1 injection. Six patients had marked or moderate relief. One patient had moderate relief after 2 injections and then complete relief 2 weeks after a subsequent neurolysis. Two patients obtained no persistent relief after the injections but had nearly complete relief of pain after later resection of a thrombosed segment of the brachial artery and a neurolysis of the median nerve respectively. In the remaining 33 patients the pain relief was inadequate and sympathectomy was considered necessary.

A compilation of the results of paravertebral injection of procaine in this entire group of 91 patients (Table 5) shows that there was complete relief of pain after 1 injection in 6 cases and complete relief within 4 weeks after

TABLE 5

Results of paravertebral injection of procaine for causalgia of the arm and leg (91 cases)

Complete Relief of Pain		Adequate but Incomplete Relief of Pain	Inadequate Relief of Pain
Immediately after 1 injection	Within 1 month		
6	7	32	46

1 to 3 injections in an additional 7 cases. Moderate relief of pain, such that further treatment was not indicated or was refused by the patient, was obtained in 32 cases. The relief of pain was inadequate in the remaining 46 patients. Sympathectomies were performed in 40 of these and in 6 operations on the nerves produced satisfactory relief of the pain.

Surgical Sympathetic Denervation of the Arm. Three types of operations were performed: a postganglionic denervation through an anterior approach, usually associated with a periarterial sympathectomy of the subclavian artery; a preganglionic denervation through the anterior approach (Telford's operation); and a preganglionic denervation through the posterior approach (Smithwick's operation). The first two types of operations were done prior to the patients' admission to this hospital. All the Smithwick type operations were done at this hospital. Seven patients were in the grade 2 group, 24 in the grade 3 group and 6 in the grade 4 group. The 37 patients can be considered together since there was no correlation between the severity of the pain and the operative results. In 2 of the patients sympathectomy was indicated to improve the circulation of the arm as well as for the relief of pain and the operations were not preceded by paravertebral injections. In the other 35 patients 1 or more injections had been done without producing adequate lasting alleviation of the pain. The temporary relief

produced by these injections was important, however, in that proof was thus obtained that the sympathectomy was indicated.

The *postganglionic procedure* was done on 21 patients at intervals varying from 10 days to 8 months after injury. There was immediate complete relief of pain in 5 cases and marked immediate relief with complete disappearance of all remaining pain during the month following the operation in an additional 5 cases. There was marked relief in 2 patients, moderate relief in 7 and slight relief in 2, with persistence of some pain in 7 of these 11 patients throughout the follow-up period of 3 to 16 months. The results of this operation were probably somewhat better than these figures would indicate since there may have been some patients who were completely relieved of pain and were able to return to duty without coming through this hospital for further convalescence. All these patients had a marked Horner's syndrome and anhydrosis of the arm, testifying to the completeness of the denervation.

TABLE 6

Results of surgical sympathectomy for causalgia of the arm (35 cases)

Type of Operation	Complete Relief of Pain			Patients with Some Persistent Pain Postoperatively
	Immediately	Within 1 month	After several months	
Postganglionic denervation	5	5	4	7
Preganglionic denervation	13	1	0	0

The *Smithwick preganglionic operation* was performed on 13 patients 7 weeks to 4 months after the injury. There was immediate complete relief of pain in 12, while the remaining patient had marked immediate relief and then complete relief during the following month. There was no recurrence of pain in any of these patients during the follow-up period of 1 to 19 months. A 14th patient was operated upon but developed repeated convulsive seizures during the operation so the procedure was abandoned and the sympathetic chain merely cut at its most accessible point, between the 2nd and 3rd thoracic ganglia. He obtained marked relief but still had some slight residual pain when he returned to duty a month later. The *Telford preganglionic operation* was done on 1 patient $2\frac{1}{2}$ months after injury with immediate complete and permanent relief of pain. The results of the two types of operations are summarized in Table 6.

In the last case in this group of 37 a preganglionic operation and a subsequent postganglionic operation both failed to give relief, but after an aneurysm of the axillary artery was excised the pain gradually completely disappeared. This patient and the one with the simple section of the sympathetic chain, mentioned above, are omitted from Table 6.

Surgical Sympathetic Denervation of the Leg. Excision of the 2nd and 3rd lumbar ganglia was done in 5 patients 2 to 5 months after injury, with the

production of marked relief in 2 and moderate relief in 3. Four of the 5 patients had some persistent residual pain on discharge from the hospital 1 to 10 months after operation. The pain in the case of the 5th patient gradually disappeared during the year after operation. In a 6th patient a high thoracic chordotomy was done $2\frac{1}{2}$ months after injury with moderate relief of pain. However, subsequent periarterial femoral sympathectomy $7\frac{1}{2}$ months after injury and lumbar sympathectomy 2 weeks later failed to lessen his residual pain.

COMMENT

In addition to the relief of pain prompt and adequate treatment of even mild cases of causalgia is important because of the rapid appearance of trophic changes when the pain is allowed to persist. Paravertebral injections of procaine are valuable both diagnostically and therapeutically and can be done early. If satisfactory injections produce temporary cessation of the pain and if adequate relief of the pain is not obtained within a few weeks, sympathectomy should be done as soon as the patient's general condition will permit.

A summary of the results of paravertebral injection of procaine in this series of cases shows that complete relief of pain was obtained either immediately or within 4 weeks in 14 per cent and moderate relief was produced in 35 per cent while in the remaining 51 per cent the relief of pain from the injections alone was inadequate. In this series, if 3 or 4 injections did not produce satisfactory relief of pain within a few weeks, further injections were seldom of much help.*

A consideration of the results of the sympathectomies for causalgia of the upper extremity indicated that while any type of denervation produced improvement in nearly all cases, in this series the preganglionic operations were definitely superior to the postganglionic type. The White and Smithwick dictum that "sympathectomy should be preganglionic in type and anatomically complete" seems to apply to causalgia of the arm as well as to Raynaud's disease.

On the other hand, sympathectomy for causalgia of the lower extremity did not give quite such satisfactory results, even though these operations were also preganglionic in type. We have no explanation for this discrepancy.

Surgical attention to the damaged nerves produces satisfactory relief of the pain occasionally but in the majority of the cases these operations are ineffective. In addition, the infection that accompanies so many war wounds frequently causes some delay before the nerves can be explored. A primary attack on the sympathetic nervous system usually gives one the best chance of relieving the pain and should be done as soon as possible. The required work on the nerves can then be done when the local conditions permit. The

* A preliminary report is in preparation on the use of a slowly absorbed local anesthetic agent for paravertebral injection. This promises to increase materially the percentage of cures with the use of this procedure.

treatment of the pain should not be delayed in the hope that the nerve exploration will produce a cure. Occasionally a thrombosed segment of a major artery or an aneurysm will produce a source of continuing irritation and the resulting causalgic pain may not be completely relieved until the offending structure is excised.

SUMMARY

1. The results of treatment in 100 cases of causalgia are presented.

2. The types of lesions involved are listed.

3. The time of onset, the characteristics and location of pain are discussed.

4. Diagnostic paravertebral injection of procaine was done in 91 of the patients, producing complete temporary relief of pain in 73, marked relief in 16 and no relief in 2 of the patients.

5. From the therapeutic standpoint, paravertebral injections of procaine produced complete and permanent relief of pain either immediately or within 4 weeks after the injections in 13 of the 91 patients. Moderate relief of pain was produced in 32 patients while in the remaining 46 the relief of pain produced by the injections alone was inadequate.

6. Postganglionic operations for causalgia of the arm resulted in immediate complete relief of pain in 5 cases and complete disappearance of all pain within one month after the operation in an additional 5 cases. There was gradual disappearance of the pain several months after the operation in 4 patients and in the remaining 7 patients in this group of 21 some residual pain persisted throughout the follow-up period.

7. Preganglionic operations for causalgia of the arm were done in 14 patients with immediate complete and permanent relief of pain in 13. In the remaining case in this group there was marked immediate relief followed by complete disappearance of the residual pain during the first month after the operation.

8. The results of sympathectomy for causalgia of the leg were not quite as satisfactory as for the arm. Four of the 5 patients in this group continued to have some residual pain.

CONCLUSIONS

1. Treatment directed toward the sympathetic nervous system should be instituted promptly once the diagnosis of causalgia is made.

2. Paravertebral injection of procaine is both an important diagnostic aid and a valuable therapeutic agent in the treatment of causalgia.

3. If the paravertebral injections of procaine do not result in adequate relief of pain within a few weeks, surgical sympathectomy should be done without delay.

4. The preganglionic type of denervation seems to be the operation of choice when sympathectomy is indicated for causalgia of the arm.

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TECHNICAL NOTES

A HOLDER FOR NEUROSURGICAL SUCTION TUBE AND CAUTERY

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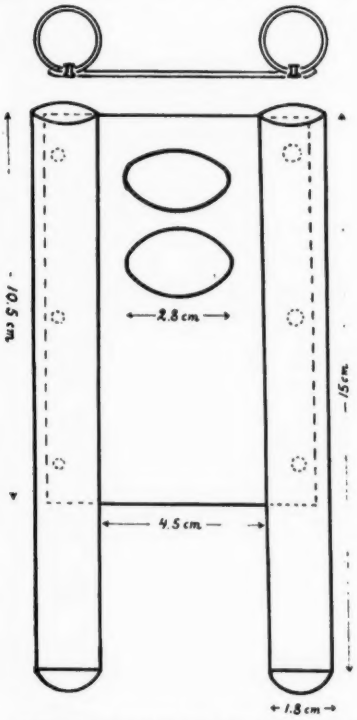
A simple holder for a suction tube and cautery, which can be attached to the sterile drapes of an operative field, has given us good service for over two years of military neurosurgery.



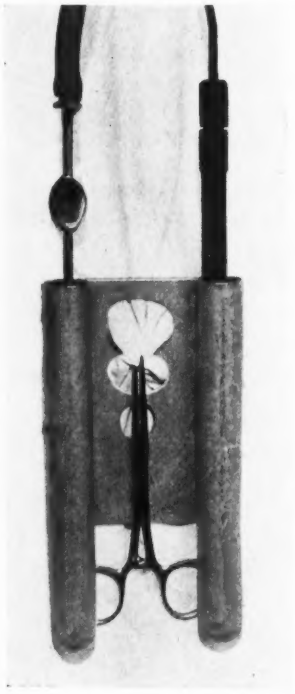


FIG. 1 (*left*). Cross section and frontal view of holder for neurosurgical suction tube and cautery. All measures are given in centimeters.

FIG. 2 (*right*). The holder hung in place, attached to a surgical drape. The tips of the drape are pulled through the two holes and clipped together with a short clamp. The handle of the clamp is pushed behind the free ends of the tubes.

It has helped to simplify our work, and since it can be easily copied in any suitable metal, we hope its detailed description may be of use to others as well.

The two most common ways to keep cautery and suction tube in easy reach of the surgeon are to fasten the cautery wire and the suction tubing to the drapes and to lay the end pieces

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of both loose on the sterile field, or to use test-tubes to keep them in place. The first method does not prevent the two instruments from slipping down and becoming contaminated; the second proved too costly, because test-tubes broke and were hard to replace. Therefore, the holder* was constructed out of aluminum (Fig. 1). To fasten it in place, one has merely to pull the sterile sheet through the two oblong holes and clip the two tips of the sheet together with a short clamp (Fig. 2). To clear the clamp from the operative field, its handle should be pushed behind the free ends of the tubes. In this fashion the holder can be hung up secure, firm, and wherever desired.

THE "SPLIT MATTRESS BED" IN THE CARE OF SPINAL CORD INJURIES

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One of the greatest problems in the treatment of spinal cord injuries is the care of decubitus ulcers and the management of the urinary catheter. Munro,[2] with his vast experience in the prevention and handling of decubitus ulcers, has emphasized the following cardinal rules: frequent turning of the patient; the maintenance of tightly drawn dry sheets beneath the skin; keeping the skin clean, massaged and dry; and the avoidance of all heretofore suggested contraptions, such as doughnuts, air mattresses, sawdust beds, Bradford frames, et cetera. Others[1] have stressed the importance of maintaining a normal plasma protein level.

Unfortunately, the vicissitudes of war with its attendant nursing shortage do not permit of such ideal care for these often forgotten patients in army and civilian hospitals. The author has observed 34 overseas returnees with complete transverse myelitis from spinal cord injuries at levels ranging from the 4th cervical to the 2nd lumbar vertebra. All but one of these patients displayed decubitus ulcers varying from 4 to 12 cm. in diameter and depth from skin maceration to bony sacrum and ilium. The sad commentary from most of these patients was that they had not been turned frequently in some stage of their convalescence, either at overseas installations or during the 2 to 7 days required for their evacuation by air. Since the patients were anesthetic below the level of the lesion they did not complain and, in fact, discouraged frequent turning. None of these patients had bed sores on the anterior surface of the body for the reason that they had not been placed in the prone position. The explanation that these patients commonly gave for this probably echoes the impressions of the profession, namely, that imperfect drainage of the bladder occurs in the face-down position because of the pressure of the body lying on the urinary catheter or rubber tubing.

A most satisfactory and simplified method of utilizing the valuable face-down position in cases of transverse myelitis has been developed. It may be called the "split mattress bed" and is prepared as follows (Fig. 1). Two regular thin felt mattresses are folded in the center and placed at opposite ends over the springs of any hospital bed with 8 inches' separation from each other. These are covered separately with tightly drawn sheets. Next, two firm, thin pillows, preferably of straw, are rolled tightly and placed about 6 inches apart in the groove between the mattresses. Thus a rectangular space measuring about 8×6 inches is formed in which the genitalia are suspended and the catheter permitted to descend directly between the bed springs into a bottle placed on the floor. The indwelling, suprapubic, or perineal catheter can be adapted equally well to this arrangement. Only a short rubber tube is required between the catheter and the bottle.

At first this method was born of necessity to avoid pressure on severe decubitus ulcers of the posterior and lateral aspects of the body. Trial disclosed that it was by far the best position for these patients. After initial reassurance the patients are surprised to find that they can live comfortably in the prone position. It is most gratifying that the bed sores

* Tec. 4 H. Perrin of the 94th Evacuation Hospital made the holder from parts of a German plane.

respond favorably to the absence of pressure. In this series of cases no medicinal ingredients were applied locally to the ulcers inasmuch as it was felt that the simple avoidance of pressure is the basic principle in their treatment. Upon arrival of the patients all devitalized tissue was carefully dissected away in order to favor drainage and prevent continued infection. The ulcer beds were lavaged daily of any accumulated exudate with ½ strength hydrogen peroxide or normal saline solutions. A cradle is used to prevent bedding from touching the buttocks or heels. Of all patients treated in this manner there was either complete healing with epithelization in those who remained in the hospital over 2 months or there was visible filling of the ulcers by healthy granulations in transient patients observed 5 to 10 days. The patients were unanimously enthusiastic about the method. This same principle was ideally

FIG. 1.

adapted by the author in the rail transportation of a patient 1800 miles by folding 2 Pullman mattresses. A basin placed directly at the groove beneath the mattresses and under the suspended genitalia was emptied of urine at intervals.

One observation which needs further confirmation is that decubitus ulcers did not develop on the anterior surface of the body in patients who were maintained in the face-down position for many days at a time, and shifted only momentarily for routine nursing care. Exceptions occurred in 2 patients who were severely debilitated, and had extensive lateral and posterior pelvic ulcers and marked nutritional hypoproteinemia. Restoration of their plasma proteins and alternate utilization of all 4 reclining positions led to complete healing of the decubitus ulcers on the anterior body surface of one patient and decided improvement in another before he was transferred. A 26-year-old male with complete transverse myelitis from a fracture dislocation at the midthoracic spine remained in the prone position on the split mattress bed for 10 months without the development of an ulcer on the anterior surface of his body. He left this position only for brief intervals on alternate days when he was placed on his side for attention to his catheter. In explanation, it might be conjectured that there are fewer bony prominences anteriorly. The costal cage is in a constant state of movement

and the pubis is suspended in the space formed by the split in the mattress. Thus, only the anterior superior spine of the ilium and the patella remain to exert pressure. Even the elbow is spared. If it is desired to relieve pressure from the anterior superior spines, the rolled pillows may be removed alternately so that intervals of freedom from pressure contact are possible.

Some advantages of the "split mattress bed" with the patient in the prone position are:

1. Nursing care is simplified. The patient may forget his catheter as it is out of his sight, and if leakage around the catheter occurs, it does not dampen the bedding but drips into a receptacle on the floor. When the catheter is no longer required, as in the case of the automatic bladder, the urine passes through the split in the bedding into the receptacle under the bed. In this instance it is wise to remove a few underlying bed spring wires. The apprehension of catching the urine in the urinal to avoid soiling the bedding is thus eliminated.

2. Involuntary defecation is managed without soiling the bed because the feces is easily collected from the crease of the upward-faced buttocks. Enemas are satisfactorily given to the patient in the prone position.

3. Of all the recumbent body positions, bladder drainage is the most satisfactory in the face-down position because of the effect of gravity aided by the dependent location of the catheter orifice. When the split mattress method is used there is no pressure of the body on rubber tubing to impede the free flow of urine. Urinary stagnation is thus minimized.

4. If it is desired to alter the patient's position even in the presence of decubitus ulcers on the hip or sacrum, this may be accomplished by lying on the side with the area of the ulceration suspended in the mattress space or on the sacrum in like manner. This avoids pressure on these areas but is not recommended for prolonged periods because of the rubber-doughnut effect which is created.

5. Foot-drop is avoided by either suspending the foot over the end of the mattress or by placing a pillow beneath the legs with the knees slightly flexed.

6. Mild hyperextension of the spine is maintained and this may be increased by supports placed beneath the chest.

7. The patient in the prone position will instinctively move his arms and shoulders frequently to change the position of his head, chest and upper extremities. In this manner the patient unconsciously exercises his arms and shoulder girdle muscles far more than he would by lying passively on his back or side, and in fact probably exercises more in 24 hours than he would by the sporadic use of dumb-bells and overhanging bars. Patients who have spent many months in the face-down position support themselves on their elbows for hours while reading, with the head held as high as one foot above the mattress. A healthy individual unaccustomed to this would find it difficult to do for more than 15 minutes. The importance of this detail lies in the fact that the development of many of these same muscles is essential in the successful utilization of crutches and braces in their rehabilitation program. Further, alternating the head from side to side aids in preventing stiffening of the cervical spine.

The patient who is placed in the face-down position will find variation by shifting a soft pillow beneath his chest and a small pillow about his head. Eventually, however, he will find that a small pillow for his rotated head, or no pillow at all, is the most comfortable arrangement. Patients may comfortably eat, read, and sleep in the prone position. Initial insistence by the physician and nurses during the first few days that the prone position be maintained will usually be necessary until the patient realizes that it is to his advantage and comfort to live face down.

SUMMARY

1) The "split mattress bed" may be readily constructed of equipment available in any hospital.

2) With the patient lying prone on this bed, pressure on the hips and sacrum is obviated while urinary drainage is encouraged and simplified.

3) Frequent turning of the patient is desirable in the prevention and treatment of bedsores in cases of spinal cord injury. When this is impossible, however, the prone position can best be tolerated for prolonged periods by the patient, inasmuch as decubitus ulcers are less apt to develop on the anterior surface of the body.
4) Nursing care of patients with transverse myelitis is greatly simplified by the use of the "split mattress bed."

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BOOK REVIEW

THE CARE OF THE NEUROSURGICAL PATIENT, BEFORE, DURING AND AFTER OPERATION.

By Ernest Sachs. *St. Louis: C. V. Mosby Co.*, 1945, 268 pp.

In his recent monograph on "The care of the neurosurgical patient" Dr. Sachs has written an exceedingly informative and well considered treatise on a subject that cannot have too much emphasis. To those who have not had Dr. Sachs' long experience in the minutiae of the pre- and postoperative care of neurosurgical patients the book should have particular value and will undoubtedly give most helpful advice in situations that might be unfamiliar. But even the more experienced will do well to peruse these pages carefully if for nothing more than to remind themselves as to their own shortcomings and perhaps to receive a needed impetus for greater efforts along the lines he has indicated.

The book is a splendid exposition of neurosurgical methods and practices as carried out in one of the best neurosurgical clinics in this or any other country, by one who has lived and worked through the infancy of this specialty and who has contributed largely to the growth and improvement of these methods and practices throughout his distinguished career.

There are no particular "high points" that should be dwelt on at length in a review of this kind, since the whole book is a mine of information as to the details of caring for neurosurgical patients from the time they are first seen, through their operative course and their postoperative care. Sachs has stressed, as did Bergman and Cushing, the importance of the neurosurgeon making his own diagnosis and thus taking the responsibility which must be his for any operative procedure advised. He discusses the uses and dangers of lumbar puncture and it would be well for all those who may be called upon to use this procedure to pay heed to his remarks. Its uses are manifold but its dangers under circumstances of increased intracranial pressure have never yet been appreciated generally and unnecessary deaths are still occurring because certain facts are misunderstood or ignored.

In Chapter One the importance of what appear to be purely scalp wounds is stressed. This is a matter that bears special mention in view of the large number of head injuries which occur every year throughout the country. It is a tragedy when an underlying serious brain injury is overlooked by insufficient investigation of what appears to be a trivial wound. This point has been emphasized by others also, particularly as a result of war experiences.

In his chapter on operating room set-up Sachs says that he prefers white gowns rather than the grey or grey-blue which are now commonly used in many neurosurgical clinics. This, of course, is an individual preference, but will doubtless come as somewhat of a surprise to those who have become used to the darker shades, as the latter have been felt to give less light reflection, and thus slightly better visual perception.

As to anesthesia, avertin and novocain are preferred in nearly all instances and Sachs discusses carefully the technic of using this combination, in particular the preparation of the avertin. To have satisfactory anesthesia, meticulous attention to details is necessary. Other neurosurgeons prefer different forms of anesthesia, such as novocain alone or intratracheal ether, according to their own experience or the circumstances under which they are obliged to work.

Chapter Four takes up the matter of certain neurosurgical methods, beginning quite naturally with the control of hemorrhage. For the scalp Sachs prefers Michel clips to the ordinary hemostats and there is much to be said for this technic inasmuch as it gets rid of certain incumbrances associated with the latter. Highly important in this chapter is the emphasis laid on the correct type of scalp incisions and the correct way of reflecting the dura after a bone flap has been turned down.

A number of illustrative clinical cases with the operative procedures carried out in them forms the basis of Chapter Five. Among many helpful suggestions here is the method of

making a complete investigation for lesions of the cerebellum or posterior fossa. Dr. Sachs likewise stresses, as would the majority of neurological surgeons, the danger of irradiating children who are suspected of having a cerebellar medulloblastoma without pathological verification of the lesion. Unless such verification is made beforehand, one can never be sure of the type of tumor or other condition present and a number of avoidable tragedies have occurred as the result of this type of promiscuous irradiation. Various methods of treating brain abscesses are dealt with, and the occasional use of the "old fashioned" subtemporal decompression in head injuries and in arachnoiditis is rightfully mentioned.

In his discussion of the treatment of trigeminal neuralgia Sachs admits of only two operative possibilities, i.e., alcohol injections and sensory root section. Many neurosurgeons, including the writer, feel that at times peripheral neurectomies, particularly supra-orbital neurectomies, under certain conditions are still extremely useful procedures. In connection with root section, it will surprise some to learn that Sachs is now using the intradural temporal approach as described by Wilkins. This procedure no doubt avoids certain vascular difficulties, but may possibly have more potential hazards.

Dr. Sachs has had a very considerable experience with choroidectomy for communicating hydrocephalus and gives an excellent description of the operation that he has found in his experience to be best suited for this abnormality. He turns down small bone flaps on either side of the head and after emptying the cerebrospinal fluid the choroid is coagulated in a dry field in order that there may be no difficulty with hemostasis. A carefully prepared artificial cerebrospinal fluid is then replaced. Another important operative technic in this chapter is the subpial cortical resection for Jacksonian epilepsy.

Spinal and peripheral nerve operations are taken up in Chapter Six. Again, the details of the various procedures are excellent, but the writer would criticize the lower inset in the drawing illustrating cordotomy. From this it would appear that little more than the anterior cerebellar tract (Gowers) was severed, and the spinothalamic tract hardly entered. In other words, the area sectioned should be shown as considerably deeper.

In operating upon lumbar intervertebral ruptured disks Sachs exposes the spines and laminae bilaterally and removes the spine of the fifth and sometimes of the fourth lumbar vertebra. It is my impression that most neurosurgeons prefer as a rule a unilateral exposure, and that the removal of spinous processes ordinarily is not necessary.

The remaining three chapters deal with the closure of wounds, the postoperative care of patients and a brief review of neurosurgery. As to postoperative care, Sachs properly emphasizes withholding anything by mouth in patients who have difficulty in swallowing. A suction apparatus should be at the bedside to remove mucus collections in the throat. This at times is a life-saving measure.

The book is profusely and carefully illustrated throughout with photographs, diagrammatic sketches and half-tones which add greatly to the appreciation of the text.

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INSTRUCTIONS FOR USING THE CUTANEOUS RESISTANCE RECORDER, OR "DERMOMETER," ON PERIPHERAL NERVE INJURIES, SYMPATHECTOMIES, AND PARAVERTEBRAL BLOCKS*

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INTRODUCTION

BASIC for the use of the dermometer is the following knowledge regarding the anatomical, physiological, and neurological factors that are involved in the electrical resistance of the skin:

(1) The resistance offered to the passage of a minute, imperceptible, direct current through the body is localized almost entirely in the skin. Thus it has been shown that a puncture made through the skin with a hypodermic needle reduces the resistance from any level practically to zero, no matter how high it was for the previously intact skin. The resistance over an abrasion or cut is likewise low.[4,10,11]

(2) Skin resistance is controlled largely through the nervous system. Thus, section of a peripheral nerve trunk produces a great and permanent increase in the resistance of the skin supplied by that nerve, while stimulation or irritation of the nerve has the opposite effect, producing a decrease in the skin resistance.[11,16,19,34,35,38,41,42]

(3) Skin resistance depends on the sympathetic component of the peripheral nerves.[3,6,7,9,12,20,23,26,27] Thus, it has been shown that: (a) Elimination of the sympathetic supply by ganglionectomy, while leaving the motor and sensory fibers intact, produces an increase in skin resistance as great as that produced by section of the entire nerve trunk.[30,31,32] (b) Transection of the spinal cord at T-1, that is, above the thoracolumbar outflow of the sympathetic nervous system, increases the skin resistance over the entire body, on the head as well as on the arms and legs; transection of the cord at L-2, that is, below the outflow, does not increase the skin resistance on any part of the body; while transection of the cord within the outflow increases the resistance of the skin supplied by the spinal cord segments below the

* Part of this work was carried out under a contract, recommended by the Committee on Medical Research, between the Office of Scientific Research and Development and The Johns Hopkins University, and part with the aid of a grant from the John and Mary R. Markle Foundation.

The technique described in this set of instructions was developed largely at the Walter Reed General Hospital in Washington. Work there was started with the help of Colonel R. Glen Spurling and was continued with the help of Lt. Col. Barnes Woodhall. Through their efforts the dermometer was adopted as part of the standard equipment of the Neurological Division of the Army.

level of the lesion.[21] (c) Factors stimulating the sympathetic nervous system, such as external heat, muscular effort, emotional excitement, or mental tension, tend to decrease skin resistance. The opposite is true of factors decreasing sympathetic activity, such as rest, sleep, relaxation, and freedom from mental tension. These act to increase skin resistance.[5,8,13,14,15,17,18,29]

(4) Skin resistance varies directly with sweat gland activity, becoming low when the sweat glands are active and high when they are inactive.[1,2,10,22] It must be emphasized, however, that these changes do not depend on the presence of moisture on the skin so much as on actual changes occurring in the sweat glands themselves. The fully activated sweat glands of some persons produce very little or no sweat, while those of others produce large amounts even when only partially activated. In persons who sweat readily the results of the Minor starch-iodine sweating test agree closely with skin resistance determinations, the areas of high skin resistance corresponding almost exactly with the non-sweating areas.[24]

(5) Local factors may also affect skin resistance. Thus irritation of the skin by physical or chemical stimuli, such as friction, passage of a large galvanic current, heat, or contact with acids, will decrease the resistance. Factors that tend to narcotize the skin increase the resistance.[15]

(6) Normally innervated skin shows, under ordinary conditions, distinct regions where the resistance is high and others where it is low. In general, low readings are obtained for areas with a rich sweat gland supply such as the palms of the hands, soles of the feet, center part of the face, the axillae, and the antecubital fossae.[24,25] These areas of low skin resistance have sharp boundaries, often quite as sharp as those that surround denervated areas. They change in size and shape with relation to various general physiological changes which affect the entire body. Thus, when a person is exposed to external heat, these areas enlarge and finally merge into one area of low resistance which includes the entire surface of the body, and when a person is exposed to cold external temperature, the areas gradually become constricted and finally disappear, leaving only high skin resistance. The sharp lines that bound these areas of low skin resistance may at times be mistaken for outlines of denervated areas. Heating a patient for a few minutes in a hot air cabinet or under a heat cradle quickly abolishes these lines and sharpens the outlines of the denervated areas. During spells of hot weather in the summer these patterns may become reversed so that the palmar and plantar surfaces have a higher resistance than that present on the rest of the skin. Such reversed patterns are also found in patients with severe fatigue.

(7) Scar tissue shows a very high skin resistance.

DESCRIPTION AND CARE OF SMALL PORTABLE DERMOMETER

The small portable dermometer shown in Fig. 1 is used to outline areas of high and low resistance.[35] (A more complex unit is required to obtain a quantitative rather than a relative measure of skin resistance.) The portable instrument consists of a microammeter (range 0–20 or 0–100 microamperes), a $4\frac{1}{2}$ volt battery (or three $1\frac{1}{2}$ volt batteries in series), a 1000 ohm potential divider, and a telephone plug socket furnished with a jack switch. A telephone plug

which fits into the socket on the panel of the dermometer connects the two silver-plated phosphor-bronze electrodes with the instrument. One electrode is constructed so that it can be quickly and firmly clipped to an ear lobe, the other has the shape of a hammer with a large disc at one end of the head and a small disc at the opposite end. By holding the handle the examiner can touch the flat surface of either disc to the skin on any part of the body and use the larger disc for general mapping, the smaller for detecting greater detail, particularly when working on the fingers and toes.

The current from the battery passes from one electrode to the other, completing the circuit through the body of the patient and through the microammeter. The potential divider in the circuit regulates the amount of current that flows from the batteries through the patient. With the potential fixed, the amount of current registered by the microammeter depends on the resistance offered by the patient's skin to the passage of the current. Thus, when the skin has a high resistance, little current flows, while when the skin has a low resistance the current

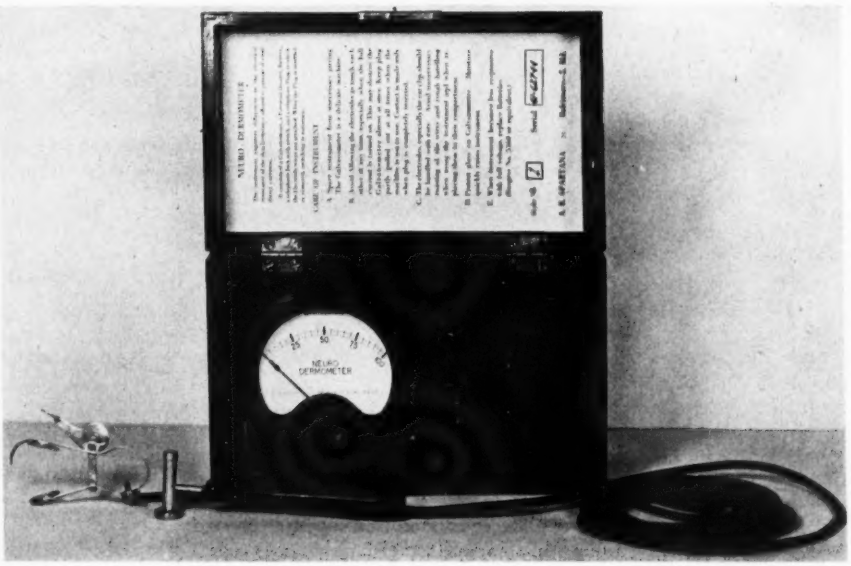

FIG. 1. Photograph of the dermometer open and ready for use.

flows freely. With a high skin resistance the pointer on the microammeter may not move at all, even when the full current from the battery is allowed to pass through the patient, while with a low skin resistance it may flick instantaneously entirely across the dial even when only a small current from the battery is allowed to pass through the patient.

Jasper has recently designed a portable instrument which can be used to obtain a measure of skin resistance as well as simply to map areas of high and low resistance.[37] An instrument described by Levine[39] which performs these same functions has long been in use in this laboratory. However, it is too large and delicate to be carried around easily on the wards. It has the advantage, however, that with its use skin resistance can be read directly from the ohmmeter and does not have to be read off of calibrated scales.

By using four $4\frac{1}{2}$ volt batteries, instead of only one $4\frac{1}{2}$ volt battery in our instrument, its range of usefulness can be considerably increased. Such an instrument was used in mapping

denervated areas in monkeys, which, in general, have a much higher skin resistance than is found in human beings.[35]

PREPARATION OF THE PATIENT

To obtain sharp outlines of the denervated areas of the skin (i.e., areas with high resistance), the resistance of the skin of the contiguous normally innervated areas must be as low as possible with relation to the level of resistance on the denervated areas. The most simple and effective method of lowering the resistance on the normally innervated areas is to expose the patient to external heat for a few minutes, either in a hot-air cabinet or in a heat cradle. This heat has little or no effect on the resistance of denervated areas of skin. Systemic measures, such as administration of aspirin and hot tea, may also be used, either separately or with the heat cradle or hot-air cabinet. In a small percentage of patients these different methods, used either separately or in combination, do not suffice to cause sweating or show a really low skin resistance on the legs.

Heating in a hot-air cabinet or cradle has the additional aforementioned advantage that areas of low skin resistance which are recorded in normal individuals at room temperature (on the face, hands, feet, and in the axillae) no longer appear after heating, as the resistance is made uniformly low all over the body. Heating also helps to make the sensory reports of the patients more accurate. Whenever possible it is well to examine the patient while he is still in the cabinet and is being heated.

Injection of pilocarpine as a means of lowering normal skin resistance does not give constant outlines of areas denervated by peripheral nerve lesions or sympathectomies. Adequate information is not yet at hand as to the constancy of outlines obtained with furmethide.

STEPS INVOLVED IN OUTLINING AREAS OF SKIN DENERVATED BY PERIPHERAL NERVE INJURIES

(1) Cleanse ear lobe with alcohol and prick skin with hypodermic needle to eliminate skin resistance.

(2) Place paste (kaolin and saturated zinc sulphate solution) or electrode jelly on disc of clip electrode, fastening electrode to ear lobe.

(3) Insert telephone plug its full length into socket on instrument panel.

(4) Turn knob that controls potential divider as far in clockwise direction as possible.

(5) Place dermometer in position so that the microammeter dial is readily visible at all times.

(6) Explore skin distal to injury with hammer electrode without paste or jelly, always keeping all of the flat surface of the electrode in contact with the skin.

(7) Watch pointer on dial while flat surface of larger disc is touched to skin:

Sharp excursions entirely across dial	= Normally innervated skin
No response	= Completely denervated skin
Intermediate response	= Partially innervated areas or normally innervated skin in persons who cannot be made to sweat—found usually on the legs

(8) When area is found that gives no response, test symmetrical area on other side of body to make certain that it gives a normal response. Repeat checks frequently throughout examination.

(9) Start to outline areas of high skin resistance. Slide larger disc of electrode over skin from area that gives no response toward area that gives very active response. (Never go in reverse direction, as this may drag moisture over denervated area.)

(10) Observe when pointer gives first small response; mark position of leading edge of disc on skin with black skin pencil.

(11) Repeat same process in successively adjoining regions until finally marks can be joined together with black pencil to form a continuous line. Entire area of high skin resistance has then been outlined.

(12) Move electrode from area of high resistance across black line and observe when response on dial suddenly changes from response of few divisions to full normal response. Mark this point with black pencil. Repeat in successively adjoining regions as before until entire "zone area" of intermediately high resistance has been outlined.

(13) Reexplore region surrounding area of high skin resistance, especially toward scars, to make certain no other areas of high skin resistance have been missed. In some patients several small islands of high resistance separated only by a fraction of an inch may constitute the denervated areas.

(14) If during the original exploration of the skin no pattern of high skin resistance can be found, then reduce the current and reexplore. Continue to reduce the current until the instrument gives only a small response over normally innervated areas. If then no patterns are found it can be concluded the skin has its normal sympathetic innervation.

(15) In some patients the previously denervated areas may give a larger response than that found on the normally innervated skin. Here, again, checks should be made with symmetrical areas on the opposite extremity. Such areas may be brought out better by placing patient in a cold room.

(16) Immediately on completing the skin resistance examination the telephone plug should be partly removed to break the current and thus to protect the microammeter in case the two electrodes should come into contact.

(17) Start sensory examination preferably while patient is still in cabinet and well warmed. The heat greatly aids the accuracy of the sensory examination.

(18) Blindfold patient to prevent him from being influenced in his sensory reports by seeing the skin resistance lines.

(19) Outline areas of anesthesia. Compare with skin resistance outline. There is usually very close agreement between areas of anesthesia and areas of very high skin resistance. When discrepancies occur check skin resistance markings again.

(20) Outline areas of dullness. These areas usually agree closely with areas of intermediately high skin resistance. Check as before.

(21) When tests are completed remove telephone plug from panel board and then disengage ear clip.

(22) Copy outlines of skin resistance and sensory pattern on body charts specially provided for this purpose.

(23) Photograph, if possible.

(24) When desirable to provide a check for future examination, inject methylene blue under the skin at key points of outlines.

CHARTS USED FOR RECORDING SKIN RESISTANCE PATTERNS

For recording the denervated areas of skin, charts should be used that show every part of the areas.[40] Charts that show only front and rear views of the extremities in most instances fail to show the parts of the arms along the lateral and medial surfaces—in other words, they have large "blind" areas. Skin resistance mapping emphasizes the importance of the continuity of outlines, that is, complete patterns. Special charts were therefore devised for this work. In order to facilitate the process of copying the patterns of high skin resistance from the body to these charts, the extremities are drawn in postures that can be most easily and comfortably maintained by men crippled with peripheral nerve injuries; further, all fixed points, bony prominences, which can be used to orient patterns are marked.

Fig. 2 shows the chart for the right arm. The two drawings in the lower center of the chart show the palmar and dorsal views of the hand with the forearm bent at the elbow. This position of the arm can be readily held by most patients with peripheral nerve injuries. These two views, however, do not show the parts of the forearm and hand along the radial and ulnar

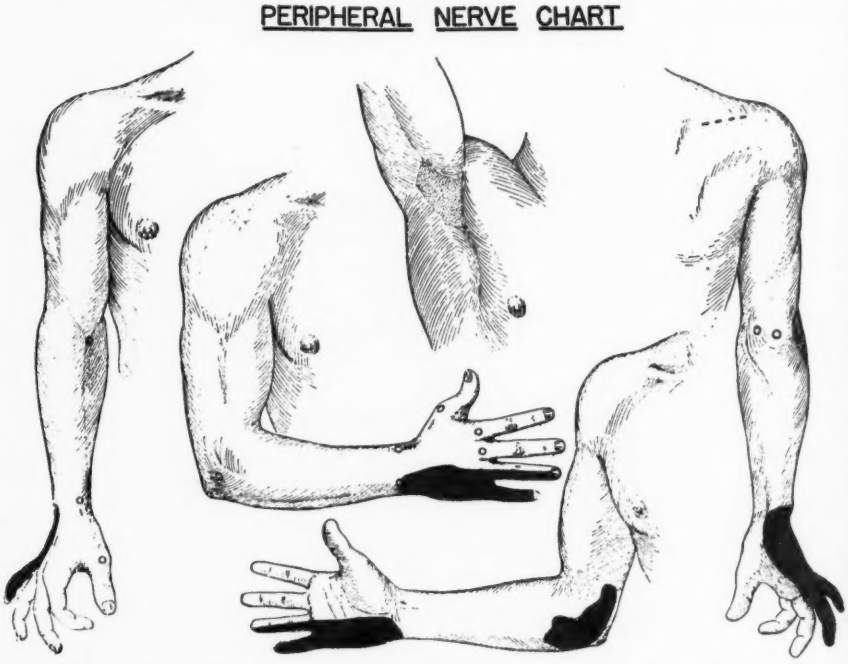

FIG. 2. Peripheral nerve chart for the right upper extremity and areas of high electrical skin resistance, or denervated areas (stippled), produced by lesion of the ulnar nerve at the elbow.

surfaces, or of the upper arm along the anterior and dorsal surfaces. These areas are shown in the drawings at either side, which show the arm hanging freely at the side of the body with the palmar surface of the hand turned toward the body. The medial and lateral views of the fingers are shown as well. The drawing at the top center of the chart shows the axillary region.

This particular chart gives the record of a soldier who was injured by a shell fragment in the lower arm and elbow in November, 1943. The cut ulnar nerve was sutured at the elbow in May, 1944. This record was taken in July, 1944. The stippled areas show high electrical skin resistance. One area included the usual ulnar field; the other the region around the scar.

Fig. 3 shows a chart for the left arm. These charts for the right and left arms are usually printed on the front and back of one sheet. This chart shows the record of a soldier who received extensive injuries about the shoulder

with evulsion of the roots from the spinal cord at C-5 and C-6 leaving C-7 and C-8 intact but compressed by edema. The wound was explored in December, 1943, and again in May, 1944. This record was taken in July, 1944. The area of high electrical skin resistance included almost all of the area usually supplied by the brachial plexus.

Fig. 4 shows the chart that is used for the legs. The drawings at the upper left show the front, rear, and side views of the legs, while the drawing at the upper right, of the squatting position, shows the pubic region and the inner

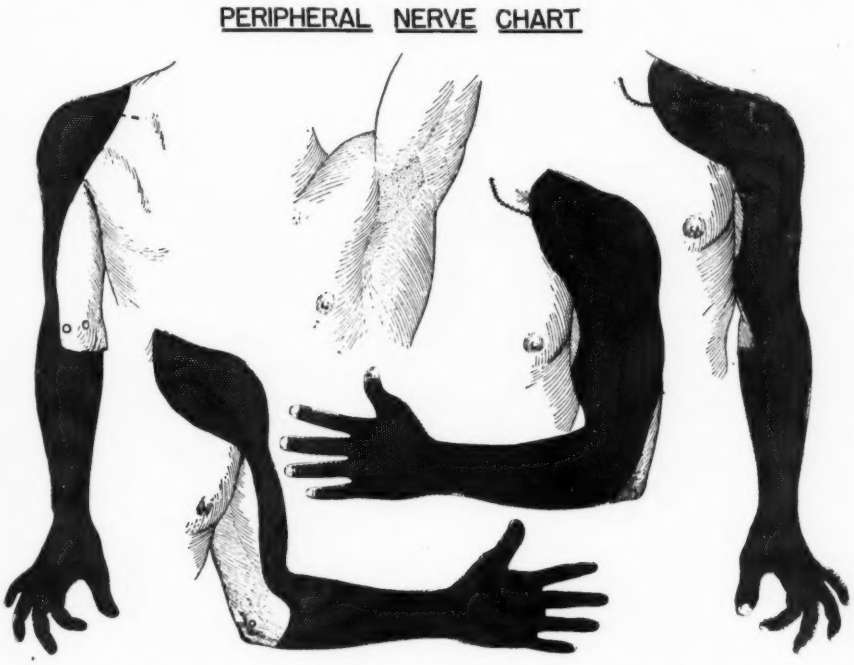

FIG. 3. Peripheral nerve chart for the left arm and area of high electrical skin resistance, or denervated area (stippled), produced by evulsion of the spinal cord roots at C-5 and C-6 and compression of roots C-7 and C-8.

surfaces of the legs. Below, the drawings show the plantar surfaces of the feet and enlarged views of the plantar and dorsal surfaces of the toes. Here, again, the charts show all parts of the lower extremities.

This particular chart gives the record of a soldier from whom on July 12, 1943, a neuroma was removed which occupied the entire cross section of the sciatic nerve. At the same time the sciatic nerve was sutured. Eight days later this record was taken, which shows a typical sciatic pattern.

Fig. 5 shows one of the charts that is used to record the changes of patterns that occur with regeneration of the nerves. In such a chart the skin

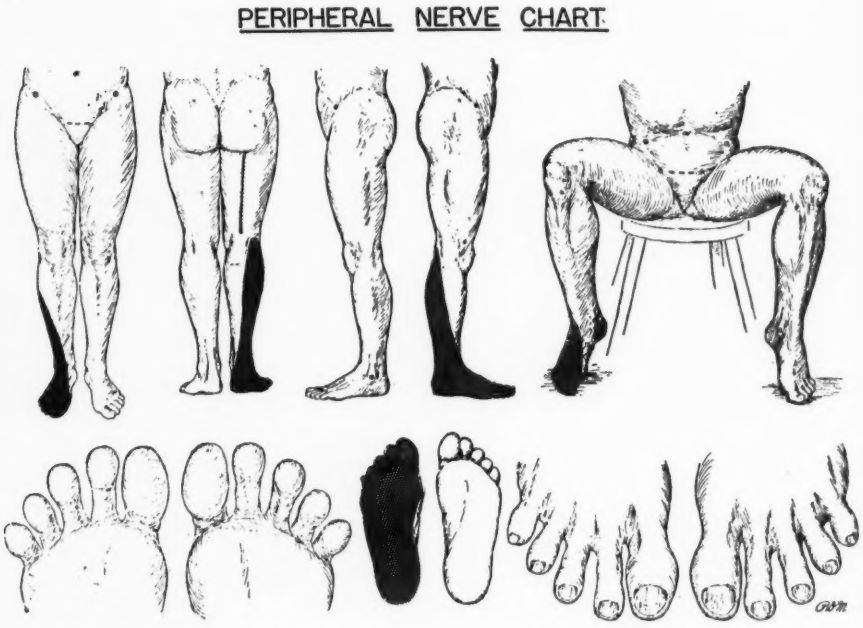

FIG. 4. Peripheral nerve chart for the legs and areas of high electrical skin resistance, or denervated areas, produced by section of the sciatic nerve.

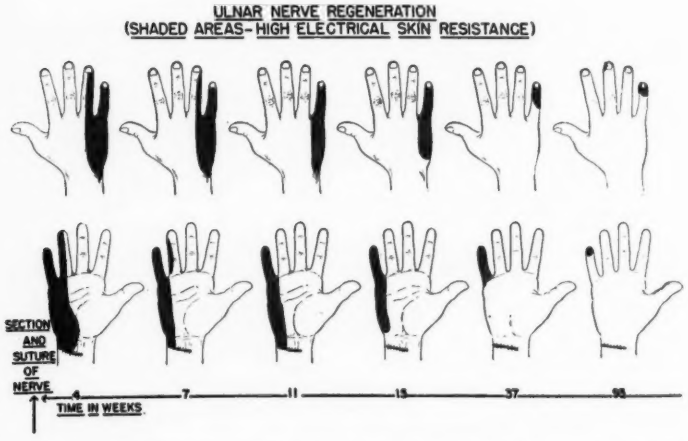

FIG. 5. Record of a patient with section and suture of the ulnar nerve, showing changes in pattern of high electrical skin resistance associated with regeneration of the nerve.

resistance patterns of the dorsal and palmar surfaces of the hands can be recorded at various intervals after the section and suture of the nerve. Charts of such a series of hands, or of arms, feet, or legs come either in printed form or can be constructed by the use of rubber stamps for the different parts of the extremities.

This particular record shows the changes in the ulnar patterns that occurred in a patient in whom the nerve had been cut accidentally at the wrist and then had been sutured within 24 hours. Four weeks after operation the patient still showed the full ulnar pattern that was present immediately after the nerve was cut. At 7 weeks the pattern had definitely decreased in width, especially on the palmar surface. Successive patterns show a gradual shrinkage, first in width, then in length, until finally, at the end of the 93rd week, only a small area of high resistance on the dorsal and palmar surfaces at the tip of the little finger remained.

USE OF THE METHOD TO OUTLINE AREAS DENERVATED BY SYMPATHECTOMY OR PARAVERTEBRAL BLOCKS

The technique used to map areas denervated by sympathectomy is essentially the same as that which has just been described for use with peripheral nerve injuries, except that to obtain sharp outlines it may be even more important to have the patient well heated. For some days after sympathectomy some patients may show abnormally low rather than high skin resistance patterns. The most reliable records are obtained 10 to 18 days postoperatively.

To map areas of skin affected by paravertebral blocks the patients should, if possible, be kept for half an hour or longer beforehand in a well-warmed room, covered with blankets. Immediately before the injections of an anesthetic agent are made the skin on the entire body should show a fairly low resistance. Under these conditions the areas affected by blocks can often be sharply mapped within a few minutes after completion of the injections.[28] Some speed and experience are required to outline and check the areas of high skin resistance before the effects of the anesthesia disappear when a short-acting agent such as procaine is used. In most instances the patterns obtained from blocks are quite as sharp as those produced by actual sympathectomy.

SUMMARY

The dermometer and the skin resistance method have the following advantages for the examination and study of peripheral nerve injuries:

(1) The denervated areas of skin can be quickly and accurately outlined without the aid or cooperation of the patient and on all parts of the body.

(2) The denervated areas have very sharp outlines which can readily be checked by independent observers.

(3) The technique and the apparatus are so simple that even untrained workers can outline most denervated areas after a few hours' experience.

(4) The previous determination of the skin resistance outlines of denervated areas greatly facilitates the sensory examination.

The following uses have been found for the dermometer:*

(1) To determine the distribution of the sympathetic component of each one of the peripheral nerves separately and in various combinations.

(2) To diagnose peripheral nerve injuries; to distinguish between complete and partial sections.

(3) To determine earliest signs of regeneration of nerve fibers.

(4) To determine the rate of regeneration of the sympathetic nerve fibers with relation to the sensory and motor nerves and the significance of deviations from the normal rates in any one of the three types of nerve fibers.

(5) To diagnose any injury or irritation of the sympathetic nervous system such as, for instance, pressure on the brachial plexus, etc.

(6) To determine the presence of actual nerve injury in suspected cases of malingering.

(7) To map areas of skin denervated by sympathectomy or procaine paravertebral blocks of the sympathetic nerves.[41,42]

(8) To help localize the level of injury of the spinal cord or roots.

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* Hyman and Beswick have recently reported several additional uses for the dermometer, particularly for work with Service men.[36]

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ELECTROSPINOGRAM (ESG)

SPINAL CORD ACTION POTENTIALS RECORDED FROM A PARAPLEGIC PATIENT

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SPINAL cord action potentials were recorded from below the level of proven complete traumatic transection in a paraplegic patient without exposing the cord. A three-channel ink-writing amplifier was used, ordinarily employed for routine electroencephalography. Fine insulated unipolar and bipolar needle electrodes were used to obtain readings from the isolated segments of the transected cord. The test was carried out in the hope of securing beneficial information regarding the mechanism of the patient's distressing mass flexor spasms. As far as can be determined this marks the first time an electrospinogram (ESG) has been made in man.

CASE HISTORY

Male, aged 27. Complete motor paralysis and sensory loss below the level of Th. 4 since gunshot wound of spine, 7 October 1944, over 12 months ago. Following an initial 3-month period of flaccid paraplegia, mass flexor spasms of the legs, and severe spastic contractions of the abdominal and back muscles began, so that finally the legs had to be tied down to enable the patient to rest or eat. On 25 September 1945 laminectomy disclosed a complete transection of the spinal cord at Th. 4. The distal and proximal stumps of the divided cord were freed of adhesions and scar tissue in the hope of relieving traction and thereby alleviating cicatricial irritation of the cord.

One month after operation the only change in neurological status was some reduction in the severity and frequency of spasms so that the legs no longer had to be tied down. It was also observed that the toes of both feet were subject to fairly frequent spontaneous flexor and extensor twitching movements which were entirely irregular as to time interval, amplitude, direction, and toes affected. At times either foot might move into a position of eversion or dorsiflexion, and sometimes twitching movements were observed in the hamstring muscles or quadriceps group simultaneous with or independent of the toe or foot contractions. These movements occurred even though the patient remained at rest for an hour or more.

On 27 October 1945 an electrospinogram was performed.

TECHNIQUE

A 3-channel Grass solenoid ink-writing amplifier was employed: calibration 6; 50 microvolts; frequency filter H 2. The paper speed at first was 30 mm. per second, and during the latter part of the test 60 mm. per second. The patient was placed on his left side on a comfortable bed in a shielded room. His legs assumed a neutral position in semiflexion. The indwelling catheter was clamped off after first emptying the bladder. No medications were administered. Throughout the test the patient remained quiet and cooperative. He experienced no untoward effects during or following the recording.

The back was prepared with alcohol and then merthiolate solution before being draped with sterile towels. Three lumbar puncture needles were introduced below the level of his injury: One between the spinous processes of Th. 6 and 7; a second between Th. 8 and 9; and

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a third between Th. 10 and 11. The first two barely penetrated the ligamentum flavum so as to impinge on the spinal cord. The lowermost or third needle touched the lamina of Th. 11, where it was allowed to remain, extraspinally. Through the first and third lumbar puncture needles a fine insulated wire electrode was passed; approximately 1 mm. of its tip was scraped free of insulation. Through the second or middle needle at Th. 8–9, a bipolar coaxial electrode was passed, its tip being bared of insulation for nearly 1 mm. to provide one contact, and its shield being bared for approximately 1 mm. a millimeter above its tip, thus providing a second, independent contact. All three electrodes were tested for perfection of conductivity and insulation both before and after use, and were sterilized in Bard-Parker's solution for 2 hours before being rinsed in 70 per cent alcohol and dried. All electrical connections were securely soldered.

A small shielded make-and-break stimulator was constructed, supplied by flashlight batteries and equipped with a potentiometer. No visible muscle contractions were ever associated with its use, when connected to any of the three electrodes, although alterations were induced in the ESG tracings. The ESG leads were numbered as follows:

Lead 1—Unipolar electrode at Th. 6–7.
Lead 2—Tip of bipolar coaxial at Th. 8–9.
Lead 3—Shaft bipolar coaxial at Th. 8–9.
Lead 4—Unipolar electrode at Th. 10–11 (resting on bone).

Further Data. The use of a ground electrode taped to the right ear lobe did not significantly alter the character of the ESG tracings. While electrocardiograph tracings appear in the records, they did not seriously interfere with the ESG. The selected tracings presented in Figs. 1, 2, and 3 were cut from the standard ruled electroencephalograph paper on which the records of the ESG were made. For purposes of publication each strip was reduced from its original length (representing 10 seconds time interval) to exactly 10 inches, representing 8.5 seconds at a paper speed of 30 mm. per second.

While certain striking recordings were obtained, it should be emphasized that it was impossible to know from what part of the cord, at Th. 6–7 and Th. 8–9, the readings were derived. However, control readings were made before and after the ESG was taken, with all three recording electrodes: (1) Resting against bone; (2) only halfway through the respective lumbar puncture needles, while the latter were in place as described; (3) in skin or paravertebral muscle at corresponding levels of the back. Movement and respiratory artifacts were likewise checked; and finally paraspinal skin and muscle recordings from two other patients were made with the same technique and electrodes. During none of these control tests did tracings appear in any way resembling those obtained during the ESG reported herewith. All indications pointed to the spinal cord as the source of the ESG.

RESULTS

(A) Figs. 1 and 2 represent parts of an almost continuous crescendo burst of activity lasting approximately 5 minutes, which showed fluctuations of fading activity towards its termination. This burst began 45 minutes after the start of the ESG and occurred while the patient was resting quietly with no visible evidence of muscular contractions other than respiratory. The latter showed no variation in frequency or amplitude, nor did the heart rate.

Two similar bursts occurred subsequently with different combinations of leads after the two upper leads had been advanced 1 mm. The ground lead was connected to the amplifier during the first burst but not during the two subsequent bursts.

The only evident common factor preceding all three bursts was the fact that the lower extremities were stimulated 6 or more times at intervals of 30 to 90 seconds, by touching the skin, stroking the sole of one foot, or passively bending the toes or flicking a toe of one foot. Almost every stimulus of this kind elicited a flexor withdrawal spasm of the affected extremity. With strong stimuli, such as toe bending, both extremities withdrew and contractions were observed in the lower back and abdominal muscles also. During the ESG bursts described, however, there was no visible muscle activity in the extremities, back or abdomen.

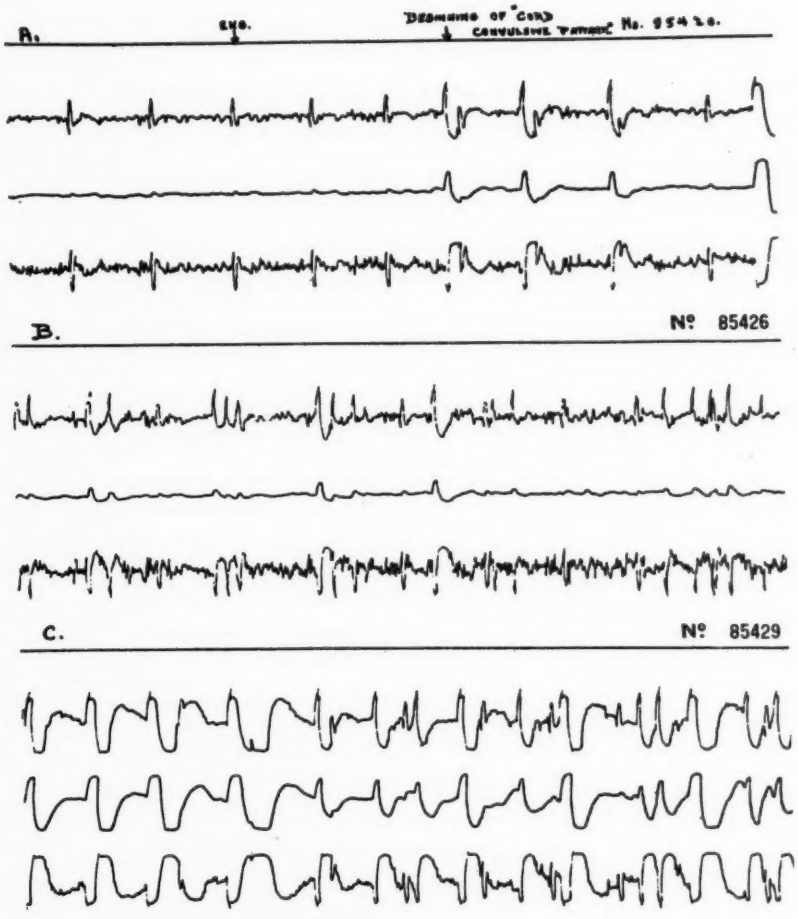

FIG. 1. Electrospinogram (ESG): Cal. 6; 50 mv.; freq. filter H 2; paper speed 30 mm. per sec.; each strip represents 8.5 sec. Heart rate 66 per min.; patient quiet. In strips A, B, C the uppermost tracing represents leads 1–2; the middle, 2–3 (coaxial electrode); and the lowest, 3–1. (A) In the 1st half is seen the base line pattern, including EKG potentials. In the 2nd half, the 5 min. crescendo burst begins ("cord convulsive pattern"). (B) Faster activity with higher voltage in top and bottom leads; 60 sec. after onset. (C) Slow wave forms and beginning of 2–3 per sec. spikes; 90 sec. after onset of burst.

Study of the recordings shows that the waves and spikes that constitute these bursts bear no relation to the EKG pattern; that they are characterized by increasingly fast activity and spikes of higher voltage than that seen in the base line tracings obtained before as well as after the burst; that large 1 to 2 per second waves occur towards the middle of the discharge, apparently synchronous in all three leads; and finally that these waves are followed by 3 per second spike and wave forms as seen in strips D and E (Fig. 2) until, in irregular fashion not illustrated, the original base line pattern is restored.

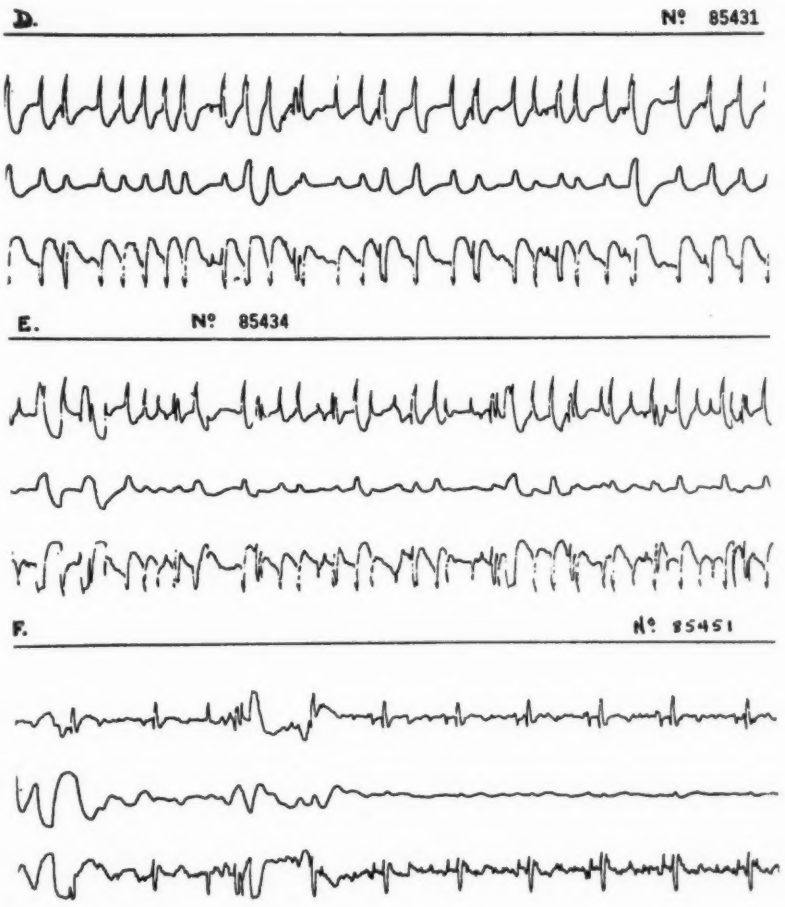

FIG. 2. Continuation of Fig. 1. (D) 3 per sec. spikes predominate in upper tracing, and 3 per sec. wave forms in lower tracing; 110 sec. after onset. (E) Beginning diminuendo of burst; 140 sec. after onset. (F) End of discharge and resumption of original basal pattern 5 min. after onset. Brief flashes of basal pattern reappeared as the synchronous discharge became increasingly disorganized, during the last 120 sec. (not illustrated).

In strips A through F the upper tracing represents lead 1–2; the middle, 2–3 from the coaxial electrode; while the bottom line represents lead 3–1.

(B) Turning now to the records of Fig. 3, in all of which the paper speed has been changed from 30 mm. to 60 mm. per second, a different type of electrical activity is seen. The two upper leads have been advanced 3 mm. beyond their position in Figs. 1 and 2. The upper tracing in each strip represents lead 1–3; the lower 3–1. Leads 2 and 4 have been disconnected from the amplifier and connected instead to the make-and-break direct current stimulator already described. The middle strip (H) is characteristic of the prevailing base line record before and after each of the bursts shown in the top strip (G) and the lower strip (I).

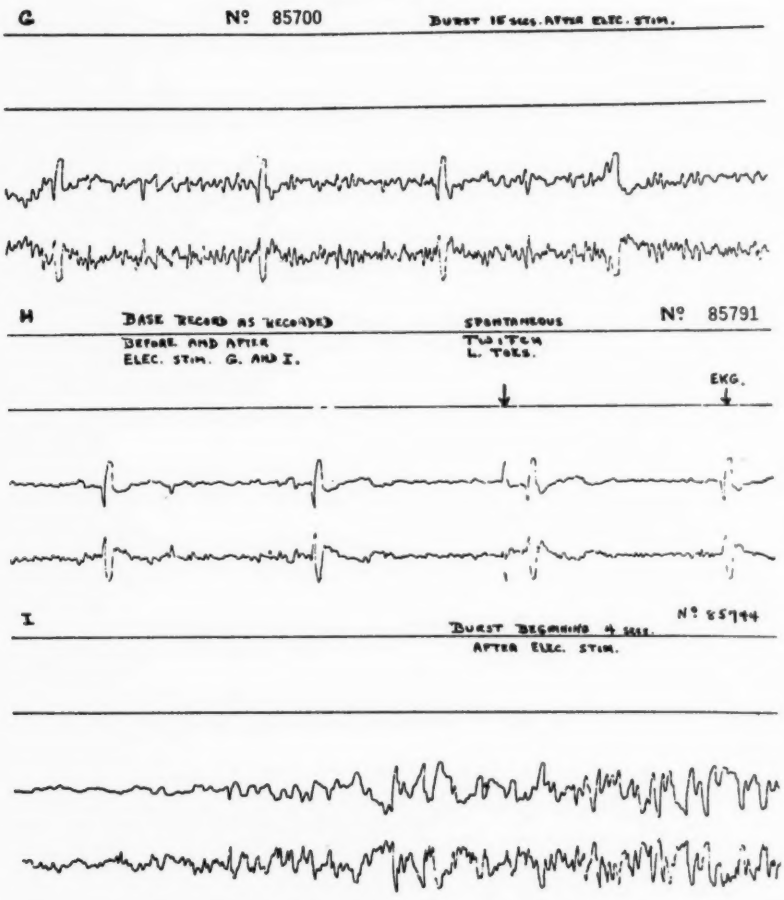

FIG. 3. ESG more than 4 min. after F. Paper speed now 60 mm. per sec. Upper tracing from leads 1–3; lower from 3–1. (G) Burst 15 sec. after repetitive make-and-break stimulation at 2.0 V. through leads 2 and 4. This burst appeared as soon as amplifier block disappeared (about 4 sec. after last stimulus) and lasted for 20 sec. Control tests later in this and two other patients with electrodes in skin or muscle, or free, did not yield such an effect. (H) Basal pattern before and after the various electrical stimuli shown in G and I. Besides the EKG pattern, a single spike appears which was coincident with a twitch of the left 1st and 2nd toes. (I) Burst appearing 4 sec. after single make-and-break shock at 2.0 V. through leads 2 and 4. Possibly (like G) an example of "after-discharge."

Near the center of H a single spike appears, obviously of fast activity, which was coincident with a spontaneous twitch of the first and second left toes, not sufficient to produce a movement artifact. Similar spikes sometimes appeared elsewhere in the record, likewise coincident with twitching movements of the toes or feet. Brief bursts of activity similar to those following electrical stimulation (as in G and I) were often associated with mass reflex spasms, whether induced by peripheral stimulation or spontaneous in onset. It is felt that the ESG has recorded action potentials associated with some of these muscle reactions.

When repetitive electrical stimulation was carried out through leads 2 (anode) and 4 (cathode) at the rate of five in 10 seconds, a burst of fast activity ensued as soon as the amplifier block disappeared, lasting 20 to 40 seconds at a maximum rate of 10 to 14 per second, at a somewhat higher voltage than the activity recorded in the base line (compare strip H). Strip G shows part of such a burst 15 seconds after the last stimulus. Strip I shows part of another burst 4 seconds after stimulation. Since no motion was evident, these can hardly represent movement artifacts; and since the EKG pattern reappears (G) the activity can scarcely be considered an amplifier artifact. In sum, it would appear that these bursts reflect an "after discharge," of spinal cord origin.

DISCUSSION

Whether or not the *normal* human spinal cord yields prolonged bursts of activity such as that illustrated in Figs. 1 and 2 remains to be seen. It would scarcely seem probable, for this burst has the earmarks of an abnormal discharge manifested by a crescendo rhythmic pattern strongly reminiscent of those recorded from the cerebral cortex during subclinical or clinical convulsive seizures. Both from a structural and physiological viewpoint, it does not seem improbable that the isolated, pathological spinal cord may be capable of spontaneous or induced mass rhythmic discharges. If such discharges are unaccompanied by visible muscular action, they might well be termed "subclinical spinal cord convulsive patterns."

Observations of two postoperative paraplegic patients lend clinical evidence for such a thesis. In each patient there occurred a continuous state of mass clonic contractions affecting simultaneously and violently the legs, and abdominal and back muscles below the level of the cord lesion. In each case this happened within 36 hours after laminectomy where dense arachnoid adhesions and scar tissue were removed or freed from the spinal cord over a distance of at least three cord segments. During these spasms all observers agreed that the lower half of the body appeared to be in "status epilepticus," the violence of which was sufficient to force the breath out in rhythmic distressing grunts. Sodium dilantin, sodium luminal, and morphine failed to relieve one of these patients, but seemed to arrest the mass clonic contractions in the other. Sodium pentothal, 2.0 gm. intravenously, was finally required to stop the "cord seizure" in the first case, even though curare, 20 mg., which had also been previously administered, failed to.

It seems probable therefore, both on clinical grounds and on electrical evidence (ESG), that the isolated pathological spinal cord may give rise to mass synchronous discharges not unlike those associated with cortical epilepsy; i.e., spinal epilepsy. It is suggested that if further therapeutic and investigative efforts be guided by this thesis, much benefit may accrue to the paraplegic patient afflicted with distressing and sometimes hazardous mass spasms.

SUMMARY

1. Records are presented which are believed to represent electrical activity within the isolated portion of the spinal cord in a "spastic" paraplegic patient.

2. It is proposed that such recordings be called an Electrospinogram or ESG.

3. Attention is directed to the probability that subclinical as well as clinical mass discharges may occur within the spinal cord of paraplegic patients, which may represent "cord convulsions" or "spinal epilepsy."

4. ESG tracings are presented which are believed to represent post-stimulus "after-discharge" and action potentials coincident with isolated muscle twitch.

CRANIOPLASTY WITH ACRYLIC PLATES

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MAJOR JAMES E. CAMERON, M.C., A.U.S.†
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THE PURPOSE of this presentation is to record the authors' experiences in 70 cases of skull defect repaired by acrylic plates made at the time of the operation by means of an impression technic.

Any method of repairing cranial defects should achieve a satisfactory cosmetic result while minimizing difficulty of the procedure, and any material used should be of sufficient strength to assure permanency of repair while achieving satisfactory cosmetic results, and such a material should produce a minimum of foreign body reaction in the tissues. Since the advent of World War II, because of the increased incidence of cranial defects, neurosurgeons of this and other countries have searched for a material that would meet these specifications. Fulcher[1] advocated the use of tantalum plates and described their successful application in patients. Since that time, the material has been extensively employed, both in military and civilian practice.

Our work was started at Fitzsimmons General Hospital, Denver, Colorado, by Major Joseph M. Cameron, M.C., A.U.S., and the technic has been modified and improved by the authors at Fitzsimmons General Hospital and at Newton D. Baker General Hospital, Martinsburg, West Virginia. Much credit must be given to the dental departments of both hospitals for their close cooperation.

The material chosen for study was a plastic, methyl methacrylate, the polymer of methyl alpha acrylic acid esters, commonly known as acrylic. The properties of the acrylic resins have been studied extensively and are well known.[5,6] Gurdjian, Webster and Brown reported the use of an acrylic plate in one case,[2] and Kerr mentions seven cases in which he employed acrylic plates in England.[3] Experimentally, the material has been employed as observation windows in the craniums of monkeys by Shelden, Pudenz, *et al.*[7] Early tissue studies with methyl methacrylate indicate that there is practically no tissue reaction to the substance in experimental animals.[4,8]

A technic was evolved which effected satisfactory cosmetic results (Figs. 1 and 2) and the material has produced a minimum of foreign body reaction in the tissue. Based on a series of 70 cases, we have concluded that acrylic may be safely used for the repair of cranial defects.

When the work was started, it was necessary, because of technical difficulties, to perform the cranioplasty in two stages, usually on successive days, and to employ a general anaesthetic. Recognizing that valid objec-

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tions would be raised to any procedure that required reopening a wound that had been sutured 24 hours previously, we succeeded in perfecting the technic so that the cranioplasty may be performed in a single procedure. In addition, we no longer employ a general anaesthetic, but resort to procaine

FIG. 1. Front and side views demonstrating the defect in the frontal region, involving the nasal process (preoperative).

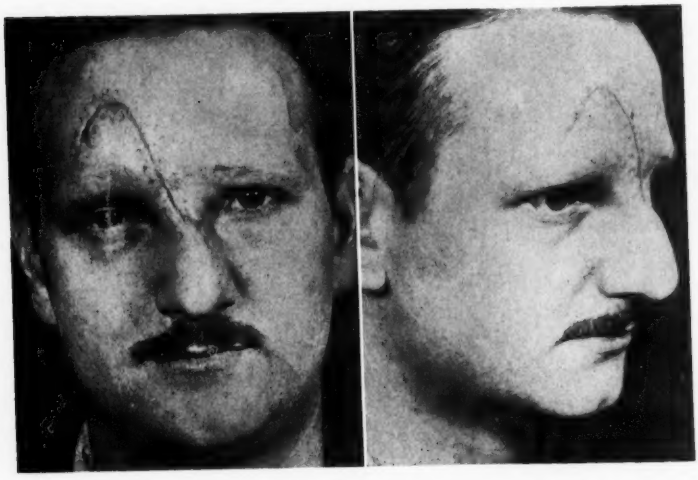

FIG. 2. Front and side views of same patient 2 weeks postoperative.

infiltration of the scalp surrounding the defect and a moderate amount of preoperative medication.

TECHNIC

When the patient is admitted to the hospital, a general physical and neurological examination is performed. Laboratory procedures include electroencephalography to determine abnormalities, particularly in the area of the defect. Following the operative procedure, the neurological examination and electroencephalography are repeated to determine any change in the status of the patient. There have been no variations between the preoperative and postoperative electroencephalograms in our patients. Variations in neurological status will be discussed later.

The patient is placed on the operating table after moderately heavy preoperative medication. The scalp is shaved widely around the defect and the field prepared with green soap and water, followed by alcohol. The line of the intended incision is then infiltrated with procaine 2 per cent, and then the drapes are applied. The incision is usually so placed as to remove as widely as feasible the old scar, although occasionally it may be advisable to prepare a fresh scalp flap. This is particularly true when dealing with large defects. The scalp edges are undercut between galea aponeurotica and periosteum around the edges of the defect and the incision is held open by means of self-retaining retractors.

In most cases, the problem that now confronts the operator is a layer of dense scar tissue which is closely adherent to the edges of the defect and which usually forms a thick false covering on top of the dura mater. Frequently we have noticed that whenever an area of dura mater had been sacrificed at a previous operation, the brain would be densely scarred and adherent to the galea aponeurotica. In order to obtain an accurate impression, the scar tissue must be assiduously dissected free from the edges of the defect (Fig. 3). The removal of scar tissue from the surface of dura mater or brain is probably advantageous from a neurological standpoint and is necessary in order to gauge the proper thickness of the plate on the impression material.

When the defect has been cleared of scar tissue and hemostatis obtained, an approximation of the outer contour of the skull in the region of the defect is obtained by placing a sterilized piece of dental base plate in the wound. Previous to this the base plate is heated in a pan of hot water until pliable. After carefully molding on the skull to the desired contour, it is removed from the incision and hardened by placing it in cold sterile water for a few seconds. The base plate must be larger than the defect, since it is to serve as an impression tray and since it determines the outer contour of the completed acrylic plate. Two retention holes are punched in the center of the tray. This is easily accomplished by using a heated blunt instrument. A layer of impression material is now spread on the inner surface of the impression tray. The material we have found most advantageous is a previously prepared mixture of beeswax and vaseline. This mixture consists of $\frac{2}{3}$ beeswax and $\frac{1}{3}$ vaseline. It may be either boiled or autoclaved to insure sterility. The impression material is firm when cool and must be softened by molding in the operator's hands. The layer of impression material which is spread on the inner surface of the tray is of sufficient thickness to obtain an accurate measurement of the depth of the bone edges. The material is pressed firmly to the inner surface of the tray, allowing some of the material to extrude through the retention holes.

An impression of the bone defect edges is now taken by reinserting the tray with its inner coating of wax into the wound and pressing down firmly (Fig. 4). The tray is then removed and the impression inspected for defects. If the operator is satisfied that the impression is satisfactory, it is placed in a pan of cold water for hardening and sent to the dental laboratory. The retractors are removed from the incision and a sterile dressing is applied to the wound, without disturbing the drapes.

In the dental laboratory, the impression and its tray are embedded in ordinary dental stone, using the lower half of a Pryor injector flask. The stone is allowed to harden and then is coated with silax. The upper half of the flask is now filled with dental stone and the halves are placed together. The lid of the flask is placed in position and the pressure plunger adjusted to the estimated top of the tray. After 5 minutes, the pressure plunger is removed and the halves of the flask are separated. The wax impression material is boiled out and the tray removed. The impression matrix is now lined with light tinfoil and packed with acrylic resin in sufficient quantity to insure a packing pressure and to allow for about 20 per cent shrinkage. The top of the injector flask and pressure plunger are reinserted and the plunger screw is adjusted until the indicator is $\frac{1}{4}$ inch above the housing. The material is cured for an average of

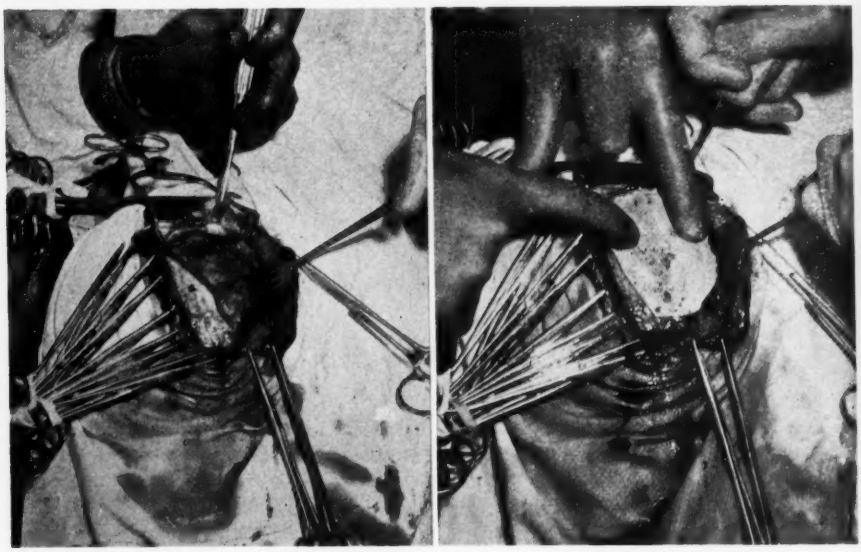

FIG. 3 (*left*). An operative field showing ridge of scar tissue which has been dissected from edges of defect (a) but prior to its removal from the surface of the dura mater.

FIG. 4 (*right*). Method of taking impression, using dental base plate as impression tray and molding to outer contour of the skull.

35 minutes by placing the flask in boiling water. During the curing process, the plunger screw will require several readjustments. After the heating process has been completed, the flask is allowed to cool for 10 minutes, after which the parts of the flask are separated and the acrylic plate is removed for polishing. In the polishing process we have found it desirable to leave a small overhanging ledge which will impinge on the outer table of the skull and prevent the plate from sinking into the defect. Several small drill holes are placed around the circumference of the plate, diagonally at the base of the overhanging ledge, to facilitate wiring the plate into position in the defect. Two large drill holes are placed in the center of the acrylic plate to insure drainage after the plate has been inserted (Fig. 5). Sterilization of the completed plate is accomplished by placing it in Bard-Parker solution for 15 minutes. The entire procedure for completing the plate requires, on the average, $1\frac{1}{2}$ hours.

When the plate has been returned to the operating room, the dressing is removed from the wound and the incision edges are again infiltrated with procaine 2 per cent. The retractors

are replaced and the plate is inserted. It should fit snugly without rocking. Small through-and-through drill holes are placed around the circumference of the bone defect and threaded with .009 mm. steel wire. Usually 4 opposing holes are sufficient to hold the plate firmly in place. These holes should correspond closely to their counterparts in the acrylic plate. The inner end of each wire is next threaded through one of the small holes previously placed around the circumference of the acrylic plate and the wires are tied tightly with a square knot. The ends are cut short and the knots are buried in the drill holes, either of the plate or of the bone (Fig. 6). Five cc. of penicillin solution containing 500 units are injected into the wound and the scalp is closed with two layers of fine silk. No drainage is employed. A fairly snug head dressing is applied and the patient returned to the ward. The time consumed for the entire procedure has averaged between 2½ and 3 hours.

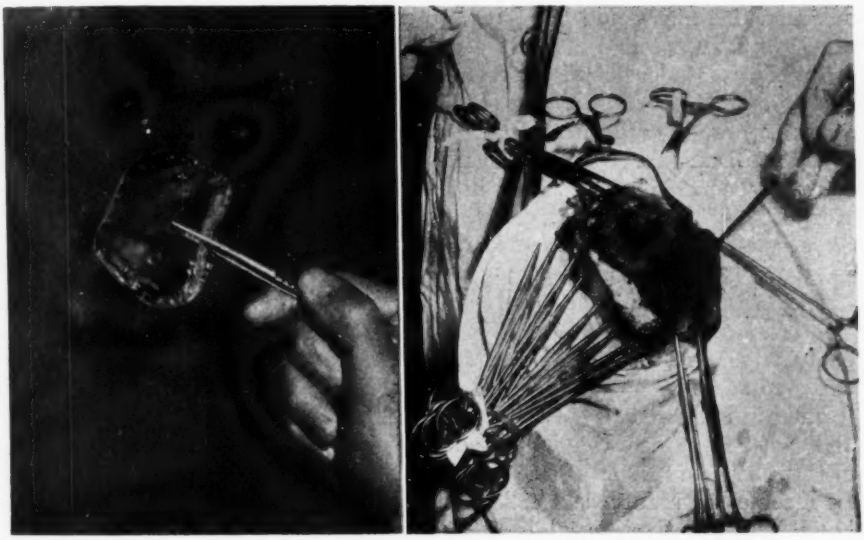

FIG. 5 (*left*). Completed acrylic plate before insertion. Drainage holes may be seen in the center of the plate and small holes for wiring are visible around the circumference of the plate.

FIG. 6 (*right*). The acrylic plate wired in place. Note the conformity to the contour of the skull and the close fit, enhanced by a small overhanging ledge on the circumference of the plate. Drainage holes are also visible.

DISCUSSION

Our series of 70 cases was compiled between June 1943 and January 1945. There were 7 infections, the first 4 of which occurred in the first 11 cases. Two of these infections occurred in cases of supposedly healed osteomyelitis of the skull. One of these healed without interference with the plate. The other required a removal of the plate because of persistent draining sinuses. This plate had been in position for 10 months and at the time of removal was essentially unchanged in appearance. One frank wound infection occurred in an uncomplicated case, but healed without interference with the

plate. The other infection was in a case of brain tumor receiving X-ray therapy. This plate was removed to facilitate disposition of the patient. In our recent cases there have been 3 slight wound infections, all of which were probably due to a necrosis in a thin scar. Each of these infections has healed without interference with the plate. From these results we have concluded that the tissues tolerate acrylic extremely well.

There have been few postoperative complications besides the infections mentioned, which could be related to either the procedure or the material used. One patient had his first convulsion 6 weeks following cranioplasty. This convulsion occurred approximately 16 weeks following the patient's injury and could conceivably be attributed to this. On a phenobarbital regime, there have been no further attacks. Two patients had mild postoperative headaches which were not incapacitating. The only other complication occurred in a patient who had had a severe head injury and at the time of his cranioplasty was suffering from post-traumatic symptoms. These were exacerbated for 48 hours. The great majority of our patients have tolerated the material very well.

As has been mentioned previously, when this work was first started, it was necessary to perform the procedure in two stages. This was mainly because of organizational difficulties. With close cooperation of both dental and operating room personnel, the procedure is now being routinely performed in one stage. This has been true for the last 43 consecutive cases. It has been noticed that the patients tolerate the procedure well and most of them are allowed out of bed within a few hours after return from surgery. In most cases a subgaleal collection of fluid develops during the first 48 hours postoperative. Simple aspiration and pressure dressings control this complication readily.

There is a tendency for methyl methacrylate to absorb water. We feel that this may be advantageous. In the first place, the amount absorbed is small, averaging .45 per cent.[5] In addition, the statement has been made that water absorption of the acrylics increases their toughness and flexibility.[6] Our first operations were done two years ago and we have noticed no complications that could be attributed to a change in the consistency of the acrylic plate and have concluded from our follow-up studies that permanent repair has been obtained. We feel that the material is satisfactory for the repair of skull defects because it is readily accessible and the plate can be made by any competent dental technician. Furthermore, the completed plate is the result of a direct impression of the defect and conforms to the defect. We also contend that there is a minimum of operative trauma. It has not been necessary, for instance, to smooth bone edges with a rongeur in order to acquire accurate fitting of the plate. This procedure would be disadvantageous when dealing with traumatic cases in which the wound was potentially infected. We suggest that the time limit between the debridement of a compound skull fracture and the insertion of an acrylic plate may be reduced. In our uncomplicated traumatic cases the average time

between injury and insertion of the acrylic plate has been 4 months. The longest time was 12 months, and the shortest time 9 days.

SUMMARY

The requirements of an ideal material for the repair of cranial defects are pointed out. A technic is described for the preparation and insertion of acrylic plates in cranial defects and the advantages of this material are discussed. Employing acrylic plates in a series of 70 cases, we have demonstrated, clinically, that there is a minimal foreign body reaction to the material and that an excellent cosmetic result can be obtained. In addition, we have perfected the technic so that the plate may be inserted at a single operative procedure with only moderate discomfort to the patient, and have decreased the operating time to within a reasonable limit. Postoperative complications are discussed and the conclusion is drawn that no permanent changes have occurred that could be related to either the material used or the method employed.

CONCLUSIONS

Methyl methacrylate is a satisfactory material for the repair of cranial defects. It is sufficiently strong to assure permanency of repair and the material produces a minimum of foreign body reaction in the tissues. Excellent cosmetic results may be obtained by an impression technic.

We have concluded that the only contraindication to the use of acrylic plates for repairing cranial defects is the history of recent infection in the area of defect. It is true that this is a contraindication to the repair of cranial defects with any material available at the present time.

No permanent postoperative complications have occurred that could be related to either methyl methacrylate or to the method described for inserting the acrylic plate.

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CONCUSSION OF THE SPINAL CORD IN BATTLE CASUALTIES

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IN CIVIL practice acute injuries of the spinal cord are almost invariably associated with dislocation or fracture of the vertebral column, and the clinical picture of transection and compression of the spinal cord from these causes is well known. In all cases of acute injury of the spinal cord—contusion, laceration, compression, transection, penetrating wounds or concussion—the phenomenon of spinal shock develops immediately and lasts for a few hours to months. According to Munro, as the result of the presence of spinal shock, "all reflex activity below the level of the injury becomes chaotic, unpredictable, variable and devoid of any diagnostic or prognostic significance."[8,9] He also emphasized the possibilities of rehabilitation in these otherwise discouraging cases by an aggressive course of good nursing, good nutrition and the use of tidal drainage of the bladder.

Transient paralysis as the result of injury of the spinal cord may occur under circumstances in which a direct blow or compression of the cord is not evident, and may be followed by partial or complete recovery in a few hours to a few months. These fortunate individuals represent spinal shock from concussion, rather than laceration or contusion of the spinal cord.

Concussion has been more thoroughly studied in regard to the brain than to the spinal cord. Experimentally it is shown to be a phenomenon consequent to sudden change of acceleration or of pressure of the brain substance.[2,3,11] It is therefore found in isolated form in the "closed" type of head injury and may be minimal or absent in cases in which there is a penetrating type of head wound. The concept that cerebral concussion is a traumatic paralysis has been challenged by Walker, Kollros and Case,[11] who, on the basis of electroencephalographic and other delicate measurements on experimental animals during the moment of impact, emphasized the excitatory phase of the reaction. It is their contention that the paralysis following injury is analogous to the depression following other types of cerebral stimulation. A similar concept of concussion in peripheral nerve, induced by an air blast, was presented by Krems and co-workers.[7] Denny-Brown[2] expressed disagreement with this viewpoint and pointed out that convulsive manifestations are rarely present in cases of cerebral concussion. It is generally agreed that synaptic transmission is affected by less intense injury than is the neuron as a whole.

The term "concussion of the spinal cord" has been used in two senses.

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Hassin[5] described as cases of concussion some in which trauma led to no immediate dysfunction of the spinal cord but was followed in a few days by symptoms and pathologic changes, sometimes simulating degenerative changes encountered in cases of amyotrophic lateral sclerosis.

In the other definition, concussion of the spinal cord is considered to be "that which happens after a single violent impact to the vertebral column, when the function of the spinal cord is affected though no gross anatomical changes can be found." We shall adhere to the second definition. While subsequent investigation may prove this erroneous, we shall presume that "concussion" is a similar phenomenon throughout the nervous system and that comparison of concussion of the spinal cord to cerebral concussion is justified.

EXPERIMENTAL CONCUSSION OF THE SPINAL CORD

The quantitative effects of measured trauma to the spinal cord were studied by Allen[1] in 1911. He made no attempt to distinguish the effects of contusion and concussion nor to evaluate the result in terms of acceleration but found that an impact of 340 gm.-cm. gave every expectation of uninterrupted recovery. By dropping a 30 gm. weight from a height of 11.5 cm. (impact of 340 gm.-cm.) onto the exposed spinal cord of dogs he produced spastic paraplegia, which largely disappeared in a week or ten days.

More recently, experimental concussion has been studied by Groat, Rambach and Windle.[4] They produced concussion in cats by a blow over the spine with a blunt wood instrument. They used the threshold stimulus of a reflex arc to determine the severity of the reaction. The degree of functional interruption and longitudinal extent of involved cord was found to be correlated with intensity of the blow applied. Immediate and delayed cytologic changes were also found.

CLINICAL OBSERVATIONS ON CONCUSSION OF THE SPINAL CORD

We have had an opportunity to observe a number of injuries of the spinal cord in an Army general hospital designated as a neurosurgical center (Lawson General Hospital). These have included transections of the spinal cord, both from battle wounds and from fracture-dislocations of the spinal column. There have also been a number of partial lesions and lesions with varying degrees of recovery. It is recognized that this case material may be artificially selected as a result of the Army casualty evacuation system but the consistency of the clinical picture lends the suggestion of validity.

Of a total of 85 cases of injuries of the spinal cord assessed, a diagnosis of true concussion of the spinal cord appeared justified in only 8 cases. These presented a similar picture, in that the lesion was caused by through-and-through (perforating) bullet wounds, which passed transversely near the spinal cord, striking bone or barely missing the lamina, and in that paralysis of the extremities persisted from a few hours to a few weeks. Holmes,[6] in the Goulstonian lectures near the beginning of the First World War, gave

the best clinical descriptions of concussion of the spinal cord that we have. The type of trauma that he found most frequently is similar to the type that we observed. He stated, "Spinal concussion is most commonly seen when the projectile has touched either a spinous or transverse process, which it may have fractured or not, but it may be also produced by a bullet which penetrates or perforates the body of a vertebra."

This is in accord with the statement of Munro[9] that "concussion of the cord is associated only with bullet wounds of the bony wall of the canal." The through-and-through nature of the wounds in cases of concussion indicates high velocity and it is probable that only in this type of injury can the "shocking" effect be produced, which will cause the requisite abrupt acceleration or compression of fluid, without actual contact with the missile or bony fragments.

In the 8 cases vesical disturbances have been absent or minimal and recovery has been more or less complete. Residual neurologic findings six months after injury have consisted in "upper motor neuron lesions," manifested by clonus, hyperactive reflexes, spasticity and presence of Babinski and Hoffmann signs. In one case in which a decubitus ulcer had been present in a region of anesthesia, this had healed and the anesthesia had disappeared. We have not encountered any cases in which a complete physiologic transection has been followed by recovery. This observation agrees with the statement of Pilcher[10] that "recovery from a complete transverse cord injury which takes place instantaneously never occurs." He emphasized that close examination of an apparently complete transverse lesion will reveal a little remaining sensation in the lower sacral distribution. However, discussion of this problem with medical officers who have seen large numbers of these cases near the combat area suggests that recovery under these circumstances may rarely occur. In looking back from the vantage point of a recovery, the best prognostic sign that can be found in the history is the minimal nature of vesical disturbance.

The following report is typical of the 8 cases of concussion of the spinal cord and is given in detail. Data on all 8 cases are given in Table 1.

Case 1. An infantry soldier, 31 years of age, was wounded in January, 1945, in Luxembourg by a sniper's rifle bullet, which perforated transversely from one side of the neck to the other at the level of the 4th cervical vertebra. Points of entrance and exit were on the lateral margins of the trapezius muscles. He had immediate paralysis in both arms and legs, associated with "numbness." He apparently did not require urethral catheterization but had persisting difficulty with constipation.

Laminectomy within 24 hours of injury showed a compound, comminuted fracture of the spinous process of the 4th cervical vertebra. The laminae were removed from the 4th and 5th cervical vertebrae. The dura was not injured and the cord was pulsating normally. A catheter was passed 10 cm. down and 7 cm. upward without encountering obstruction.

Ten days after injury, neurologic examination showed weakness of all four extremities. Reflexes of both legs were normal. Reflexes in the left arm were weak and sluggish. Cremasteric and abdominal reflexes were present. Fourteen days after injury the patient was described as using legs and feet but weakness of the arms persisted, the patient being able "almost to flex his forearm unaided." Electrical evaluation of muscles at that time showed

loss of muscle power of chiefly the median distribution on the left and the ulnar distribution on the right. One month after injury the patient was able to get his hands to his mouth to smoke. Two months after injury there was still moderate weakness, particularly of the left upper extremity.

On admission of the patient to Lawson General Hospital 3 months after injury all scars were well healed and there was no deformity or rigidity of the neck. There was no Horner's syndrome. There was disuse atrophy of all extremities, with weakness of both arms and legs. There was a bilateral Hoffmann's sign but no Babinski reflex. All deep reflexes were exaggerated. There was bilateral exhaustible clonus. Subjectively there were still regions of "numbness" on the legs and arms but objectively no anesthesia could be discovered. By 3½ months after injury the patient was able to perform skilled movements and was sent home on a convalescent furlough. Seven months after injury he was separated from the military service as having attained maximal hospital improvement and possessing good functional recovery.

TABLE 1

Recorded data of eight cases of concussion of the spinal cord

Case	Level of Injury (Vertebral)	Duration of Complete Paralysis	Duration of Vesical Distention	Fracture	Laminectomy
1	C-4	10 days	0	Spinous process	"Dura and cord normal"
2	C-7	14 days	3 wks.	Transverse process	Not done
3	C-6	8 days	0	Spinous process	Not done
4	C-7	25 days	0	0	Not done
5	T-10	14 days	0	Spine and lamina T-10 and T-11	"Cord intact"
6	T-5	14 days	0	Small defect lamina T-5	Not done
7	C-5	4–5 hours	0	Slight irregularity of spinous process on x-ray	Not done
8	T-6	7 days	1 day	Fracture body T-5	Not done

Comment on Clinical Cases. It should again be emphasized that the first 7 cases presented identical trauma with directly transverse bullet wounds just dorsal to the lamina. Patient 2 showed Horner's syndrome and had a decubitus ulcer in a region of anesthesia over the right hip, which persisted for six months. He therefore probably had a larger element of contusion than the others. Cases 5 and 6 are noteworthy in that injuries of the thoracic vertebrae were associated with paralysis in the upper extremities.

In Case 8 the lesion was a thoracic wound, which differed from the others in that the vertebral body rather than the lamina was fractured. Furthermore, there was impairment of sensation below the level of the

wound and paralysis was not present in the upper extremities. Motor function returned first in the left foot, then in the right foot, and gradually returned up to the level of injury. The recovery of sensation followed the same pattern.

Among the cases in which concussion of the cord could not be diagnosed, in 3 cases of perforating side-to-side bullet wounds there was complete paralysis without recovery. In one of these when laminectomy was performed the cord was observed to be "intact but injured." Out of 15 cases of penetrating shell wounds and 7 of penetrating bullet wounds in which there were clinically complete lesions, observation at the time of laminectomy revealed anatomic transection in 9 cases and in 5 the spinal cord was described as intact but injured. A penetrating wound is considered to be a wound with entrance, but not exit, indicating a decrease of velocity to zero.

SUMMARY AND CONCLUSIONS

In examining patients whose spinal cords were injured we have found 8 cases in which a diagnosis of concussion of the spinal cord was justified. These are similar to those previously described, particularly in the literature of World War I. Injuries to the cauda equina have not been discussed. Recovery of both motor power and sensation generally begins in the lower extremities and ascends, the duration of dysfunction being greatest in the level of injury. The exact nature of the mechanism of concussion remains obscure. It is hoped that the clinical material presented can be correlated with future experimental work.

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SENSATIONS OF ELECTRIC SHOCK ON FLEXION OF THE NECK AS A SIGN OF HEAD INJURY*

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Most of the head injuries seen in war time appear to be well localized. A penetrating bullet or piece of shrapnel may lodge in the brain tissue and consequently produce signs of local damage. However, it must be borne in mind that a blow by a missile sufficiently violent to penetrate the skull will also produce a general alteration in intracranial dynamics and neuronal function so that damage to the brain is usually much more widespread than the clinical signs would indicate. The varieties of gross and microscopical changes found in cerebral contusion and concussion have been fully described. The pathological physiology of intracranial injuries has also been amply investigated. All these studies indicate that parts of the brain far removed from the site of the main injury reveal significant alteration in the neurons and axis cylinders.[2]

Recently in a series of head injuries studied at the U. S. Naval Hospital, San Diego, California, we found 17 patients who manifested a well known sign of spinal cord disease.§ This sign, "electric-shock" or vibratory sensations (pallesthesiae) in the extremities and trunk, elicited on forward bending of the head, was found in patients with cranial trauma. The sign is part of a syndrome that has the following characteristics: the patients complain spontaneously of "electric shocks," "buzzing," "tingling," or "vibrations"; the symptoms appear from 3 to 16 weeks after the injury and may last from 2 to 20 weeks; as a rule the vibratory sensations are bilateral and symmetrical in distribution, and the radiation is along the classical dermatomes in the extremities, having a centrifugal direction toward the hands and feet. In most instances the patient is unaware of any relationship between the vibratory sensations and flexion of the head until it is demonstrated to him. The following case reports are examples of the group illustrating the syndrome:

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§ This sign was described during the last war by Marie and Chatelin in 1917[7] and Babinski in 1918.[1] And yet, strangely enough it acquired the eponym of "Lhermitte's sign."[8] Lhermitte, of course, wrote several articles on this "shocking" sensory disturbance which he found in cases of multiple sclerosis.[3,4,5,6] This subject was reviewed by Salmon in 1937.[10]

Case 1. P.E.D., a 19-year-old Marine Private, was shot through the left frontal region. The missile entered at a point above the left eyebrow and, according to x-ray findings, lodged on the right side of the brain. Except for a momentary period of unconsciousness the patient was asymptomatic. The small scar above the left eyebrow healed rapidly and the patient was returned to duty. However, 7 weeks following the injury he began to complain of headaches, rhinorrhea, and "tingling" electric-like sensations in the hands and back. The sensations, which the patient found occurred only on flexion of the neck, radiated along the mesial aspects of the arms and forearms down to the palmar aspect of the finger tips. Repeated neurological examinations were always normal. Because of increasing severity of the headaches the patient was readmitted to an overseas hospital and a decompression was performed. The electric-shock sensations lasted 2 weeks and did not recur. Subsequently he was returned from overseas and has been symptom free for many months.

Comment. This patient sustained a head injury in the frontal region and yet several weeks after the trauma he had bilateral symmetrical symptoms characteristic of spinal cord disease. The fact that the complaints appeared during the post-traumatic period and lasted 2 weeks suggests that a pathological or some dynamic change had taken place long after the injury, possibly a regenerative process. The late appearance and transient nature of these symptoms were most characteristic features of the syndrome.

Case 2. R.O., a 24-year-old Marine Pfc., was struck on the right side of his head by shrapnel. The parietal bone was fractured and a small amount of brain tissue protruded through the wound. There was paralysis with concomitant pyramidal tract signs and sensory defects on the left side. These symptoms and signs remained for more than 2 weeks. X-ray revealed a bone defect and small fragment fracture line through the right parietal bone. Four weeks following the injury the patient noticed a sense of vibration and tingling in the back which radiated forward to the chest and abdomen. The following week he noticed that these shocks extended into the anterior and medial portion of the thighs and radiated to the tips of all the toes. Three months after the injury he felt an uncomfortable wave of "electric shock" which radiated down to the fingertips along the medial aspect of the upper extremities. He also found that on occasions he could not walk well, apparently because of an inability to balance himself. It seemed to him as if his feet were "light" and unsteady. Examination revealed minimal residual signs of a right parietal lobe lesion. These, however, were overshadowed by the symptoms elicited on acute flexion of the head. On this maneuver the patient experienced disagreeable sensations of electric shock running down the medial aspect of his upper extremities into the fingertips on the palmar side and from the thighs into the legs. In this acutely flexed posture the kinesthetic sense was found to be defective and he could not recognize the vibrations of a tuning fork. These symptoms and signs lasted 4 months, gradually disappearing from the lower extremities first. X-ray studies of the spine, examinations of the spinal fluid and manometric tests were all negative. The patient compared the sensation he experienced on flexion of the head to that of a vibrating tuning fork applied to a bony prominence.

Comment. In this case the syndrome was produced by an injury in the right parietal area. Again, the symptoms were transient and appeared some time after the trauma. Significant was the absence of history of symptoms of spinal cord trauma immediately after the skull injury. The fact that sensations were first localized to the trunk, later involved the lower and finally radiated along the upper extremities as well, indicated a spreading or dynamic change within the cord.

Case 3. H.L.F., a 22-year-old Marine Corporal, sustained a shrapnel wound in the right temple. Except for strong feeling of familiarity of faces and other *déjà vu* phenomena there were no symptoms of cerebral damage. An x-ray of the skull showed a defect in the right temporal bone with small shrapnel fragments about the right temporomandibular joint. Six weeks after the injury he noticed a buzzing vibratory tingling sensation in both legs. At times these sensations radiated forward from behind the hips and down the medial aspect of the thighs. On other occasions the sensations involved only the lower torso. Examination revealed that sudden and firm acute flexion of the head produced a pronounced tingling electric-like sensation in the lower part of the body and in the buttocks. These sensations radiated anteriorly to the hips and down the medial aspect of the thighs. There was no pallesthesia in the upper extremities. However, on subsequent occasions the patient noticed that on drying his face with a towel there was a marked tingling sensation in the fingertips of both hands. An x-ray of the cervical spine was normal. Numerous neurological examinations and all laboratory investigations were found to be negative. The shocking or vibratory sensations lasted 4½ months and disappeared completely. He has been free of this symptom during the past year.

Comment. This is an example of a patient with injury to the right temporal region who showed bilateral symmetrical subjective sensory disturbance in the extremities. Characteristically, the syndrome appeared during the post-traumatic period and the duration was transient. The distribution was at first in the lower thoracic and lumbar dermatomes but later implicated the cervical segments. This again demonstrated that a dynamic process had taken place, presumably within the spinal cord.

Case 4. E.H.McI., a 22-year-old Marine Sgt., was struck by a machine gun bullet in the right side of the head. He was dazed for 15 minutes. He was returned to duty and subsequently participated in another campaign. During the post-traumatic period there developed headaches. An x-ray of the skull showed a bullet hole in the right occiput. Nine weeks after the injury he noticed a tingling sensation which started in his hips and radiated down the inner aspect of the thighs and legs to the bottom of the feet and into the toes. Three days later he noticed the same shock-like sensations shooting through the trunk and medial portions of the arms and into the palms. To quote the patient—"The sensation is the same as one has when the crazy bone is struck." He also noticed, while hiking, that bending his head forward caused him to "stagger" so that he lost his balance and often bumped into his mates. Because of this he was admitted to the hospital. His head was placed in traction and this produced some relief. The patient was subsequently returned from overseas.

At this hospital the neurological examination was negative except for a small homonymous scotoma in the left upper field of vision. Acute flexion of the neck produced electric-like sensations in the upper extremities. They no longer radiated down the lower limbs. He showed no other signs of disease of the nervous system. The symptoms of pallesthesiae lasted 4 months and gradually disappeared.

Comment. This is an example of injury to the occipital area which produced exactly the same syndrome as lesions in the frontal, parietal or temporal area. Interesting was the manner in which the distribution of the radiation of electric-shock sensations had changed.

There were several other cases of penetrating gunshot and shrapnel wounds of the brain with this syndrome. The location of the wound differed from one patient to another and some had multiple wounds. Despite the variety of lesions in the brain the syndrome was characteristically the same.

Case 5. C.E.H., a 29-year-old Marine Corporal, was rendered unconscious by the blast of an exploding shell. There was some bleeding from the left ear. Following the blast he complained of headache and defective hearing on the left. An x-ray of the skull was reported as normal. Six weeks after the concussion he began to complain of sensations of electric shock radiating down the medial aspects of the upper extremities whenever he bent forward. Acute flexion of the neck produced marked electric-shock or vibrating sensations in the hands, radiating to the tips of the fingers, and in the anterior portion of the thighs, down to the knees. During this maneuver there was no disturbance in kinesthesia. An x-ray of the cervical spine was normal, as was the neurological examination. The entire syndrome lasted over 3 months and ultimately disappeared.

Comment. This patient had the same post-traumatic syndrome as those with penetrating wounds of the head. The distribution of sensations in this case was limited to the upper extremities and was similar to Case 1. It may be questioned whether the blast of an exploding shell can produce symptoms of spinal cord damage. One cannot exclude the possibility of head or even spinal injury which the individual may have sustained during his fall as a result of the blast.

Case 6. W.H.P., a 32-year-old Seaman First Class, was struck in his head with a brick. The left temple was lacerated and the x-ray revealed a comminuted and depressed fracture of the left parietal bone. The wound was debrided and the depressed bone fragments were elevated. The immediate postoperative course was uneventful but 10 weeks after the injury he complained of tingling sensation in the buttocks and both lower extremities. Examination disclosed that flexion of the neck precipitated the symptoms to a marked degree. X-ray of the cervical spine, pneumoencephalogram and spinal fluid studies were all normal, but during his convalescence he complained of pain in the right side of the neck and the ipsilateral upper extremity. The electric-like sensations associated with neck flexion lasted 10 weeks.

Comment. This patient had a type of cranial trauma that is not infrequently seen in civil practice. Although head injuries are extremely common, the syndrome of electric-shock sensations on forward bending of the neck has not often been reported. In all cases the question of concomitant injury to the spine must be considered and in this case the fact that the patient complained of transient pain in the right side of the neck and right upper extremity suggests the possibility of such an injury. It cannot be denied that an individual who is rendered unconscious by a blow to the head may also sustain a simultaneous injury to the spine during the fall.

Walshe[12] claims that patients with closed head injuries sometimes show evidence of a lesion in the spinal cord with such signs as muscular wasting and weakness, reflex changes (depressed biceps and exaggerated triceps, hyperactive knee jerks and ankle clonus) and, in addition, alteration in the intervertebral disc space. He stressed the not uncommon incidence of minor damage to the cord in association with closed head injuries. We have found two such cases, one of which is hereby described.

Case 7. W.D.Mc., an 18-year-old apprentice seaman, ran into a jackstay and struck his forehead forcibly. He was unconscious for a few minutes. When he regained consciousness he found he could move only his head. There was paralysis below the neck. Within one week the power gradually returned except in the right arm, which recovered more slowly. Neurological examination, one week after injury, showed motor, reflex and sensory changes characteristic

of a partial lesion of the upper cervical roots on the right side. The spinal fluid examination revealed a slight increase in the total protein content and an x-ray of the cervical spine disclosed a congenital anomaly of the posterior spinous processes. The patient improved, but during the 5th week of his convalescence he complained of sensations like electric shock in his extremities, radiating distally and symmetrically along the inner aspects of the arms, and the volar aspect of the forearm, terminating in the fingertips, and along the anterior and medial aspects of the thighs, radiating to the legs as far down as the arches of the feet. These symptoms appeared on passive or active flexion of the head. The syndrome persisted for 5 weeks and during the 6th week gradually subsided. The patient ultimately improved. Except for a residual sensory defect in the right forearm, he had no complaints.

DISCUSSION

From the foregoing observations it is clear that injury to the head can produce transient symptoms during the post-traumatic period. The symptoms appear to be of spinal origin. The latter theory is based on the facts that the electric-shock sensations (a) can be elicited on flexion of the head, (b) they have a bilateral symmetrical dermatome distribution and (c) they closely resemble the syndrome found in unquestionable disease of the spinal cord. Similar observations and conclusions have been made by Reider,[9] who studied 31 such cases in a large neurological center of the U. S. Army.* Triumfov[11] reported 23 cases of intracranial injury, observed in the Russian troops, with the syndrome of pallesthesiae on forward bending of the neck.

The most characteristic feature in all of the cases herein reported is the appearance of these transient symptoms weeks or months after the head trauma. This feature is difficult to explain. Granted that a sharp blow to the cranium may produce a sudden lateral or forward movement of the head and thus cause a contrecoup injury to the spinal cord, presumably at the level of the foramen magnum, the questions that arise from this supposition are: (a) Why are they characteristically manifested as electric-shock sensations? (b)Why is it that in some cases only the upper extremities and in others the trunk and lower limbs are involved? (c) Why is it that spinal cord symptoms are not manifest immediately after the head injury? (d) Why do the symptoms appear and vary during the post-traumatic period? All of these questions are difficult to answer, but the following suppositions may partly solve some of the problems.

The available data seem to suggest that the symptoms are due to a disorder in the posterior column function.† In the cervical region the tracts that conduct proprioception from each of the upper extremities are separated by the columns that conduct deep sensibility from the trunk and lower limbs. By position in the spinal canal all of the dorsal columns appear to be

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* Although most of the patients in his series had bilateral and symmetrical electric-shock sensations on flexion of the head, there were some in whom the syndrome was limited to one or three extremities.

† The fact that the sensation elicited on forward bending of the head was often identified by the patient as the same as that perceived from a vibrating tuning fork applied to bony prominence, and the fact that defective kinesthesis, such as in Cases 2 and 4, was found during the maneuver strongly suggest that the syndrome was due to a disorder in posterior column function.

more vulnerable to injury by contiguous bony structures than any other part of the cord, especially in the upper cervical region. An anteroposterior jarring of the head might conceivably force the posterior half of the spinal cord against the edge of the foramen magnum and thus traumatize the dorsal columns. When the cranial injury produces a lateral jarring of the head, the resultant contrecoup trauma to the spinal cord might be localized more to the lateral areas of the posterior columns and thus produce symptoms only in the upper extremities.

As for absence of spinal cord symptoms immediately after the cranial trauma, Walshe[12] is of the opinion that most of the patients tend to dismiss them as natural consequences of a blow on the head and forget them, or else the physician does not elicit them in the history. We cannot agree with Walshe's opinion, because most of our patients categorically denied all symptoms of spinal cord disease prior to the appearance of the syndrome. Apparently this question cannot be answered satisfactorily at present. However, it is well known that slight or extensive damage to the nervous system may produce no symptoms. This is particularly true of tumors, and not infrequently of traumatic encephalopathy. The most puzzling feature of the syndrome is the appearance of the electric-shock or vibratory sensations later in the post-traumatic period. It is not improbable that this is the result of a regenerative process within the spinal cord at the site of the injury. This would explain the progressive changes in distribution of the sensory disturbances. Lhermitte compared the electric-shock sensations elicited on flexion of the head to the tingling noted on tapping in peripheral nerve injuries during the recovery period (Tinel's sign), thus implying that the sensations were due to a regenerative process.

Whatever the explanations, the appearance of the syndrome weeks after the head injury is in itself significant and constitutes a valuable clinical sign of trauma to the nervous system. It should aid in deciding whether we are dealing with an organic or purely psychogenic case of head injury. The occurrence of the syndrome should establish the case as being organic.

SUMMARY

In a series of 17 cases of gunshot or shrapnel wounds and other types of injuries to the head, the patients complained of sensations of electric shock or pallesthesiae in the extremities. These sensations were symmetrical, radidiated along well known anatomical dermatomes, and could be elicited on flexion of the head. They appeared during the post-traumatic period (several weeks after the injury), changed from one set of dermatomes to another, and lasted for a short time (weeks to months). It is believed that the syndrome is caused by a simultaneous contrecoup injury to the spinal cord. The occurrence of such a syndrome after a head trauma should be considered as a significant sign of injury to the nervous system and should remove existing doubts as to the organicity of the case.

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VENTRICULAR ELECTROENCEPHALOGRAPHY*

A DESCRIPTION OF THE TECHNIQUE

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THE CLINICAL usefulness of electroencephalography is limited by the fact that scalp electrodes for physical reasons record only the activity of the superficial and subadjacent tissue of the lateral and superior surfaces of the cerebral hemispheres. With present methods deep subcortical pathology cannot be detected or localized† nor can the origin of abnormal waves, such as the petit mal and psychomotor-like waves seen in epilepsy, be determined. In order to extend the clinical usefulness of electroencephalography, electrodes must be placed in or near deep subcortical structures. This has been attempted by means of a nasopharyngeal lead,[1] a procedure which is held by Jasper to be useful in the localization of lesions in the region of the third ventricle (p. 390 ff.[2]). Penfield (p. 426 ff.[2]), and Sachs, Schwartz and Kerr[3] have made isolated observations on the electrical activity of deep structures at operation,‡ and the subcortical electrical activity of animals has been widely reported. The main practical objections to the routine use of deeply placed electrodes in humans are uncertainty as to the exact location of the electrodes, and fear of damaging subcortical structures. It was thought that both these objections could be obviated by the use of electrodes placed in the lateral ventricles. Since the ventricular fluid contains electrolytes and therefore transmits electrical impulses, ventricular electrodes should record the activity of the surrounding tissue. At the same time the procedure should carry no greater risk for the patient than a ventricular tap. We are presenting our method of ventricular electroencephalography with the hope that it can be applied profitably to the study of clinical problems by investigators to whom clinical material is available.

METHOD

Electrodes consist of a 6-inch length of lacquer-insulated copper wire. The insulation is scraped off the extreme tip of the end to be inserted in the ventricle, and off approximately ½ inch of the opposite end. The wire is marked off at suitable intervals from the recording tip with white gloss-enamel paint. Before the ventricular needle is inserted, the wire is carefully measured against it so that the wire can be inserted to the same depth as the needle. When a

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† With respect to recording by scalp leads, buried cortex such as that of the insula, or deep cortex such as that of the orbital surface of the frontal lobes or the sphenoidal surface of the temporal lobes presents the same problem as deep subcortical tissue.

‡ While this paper was in press, Walter and Dovey in *Lancet* (Jan. 5, 1946, *250:* 5–9), described in detail the successful use of needle electrodes in delimiting subcortical tumors at operation, "direct electrography."

20-gauge needle is used, No. 30 wire is most convenient. No. 28 wire is suitable for use with a larger needle. After the electrodes have been prepared, they are straightened carefully. Slight twists in the wire make insertion through the needle difficult. The wires are placed in a long glass tube which is stoppered with cotton or gauze, and sterilized by autoclaving. We have always discarded such electrodes after one use.

To insert the wires, a ventricular tap is made with a dull 20-gauge needle and syringe in the usual way, using as criteria of placement the sudden cessation of resistance to injection of air and the ability to withdraw cerebrospinal fluid. In monkeys, as in infants, it is easiest to tap the anterior horn of the lateral ventricle, and most of our recordings have been made with the electrodes in this position. In a few instances, electrodes have been placed in the posterior horns. Whenever possible electrodes are placed symmetrically in both ventricles. When the

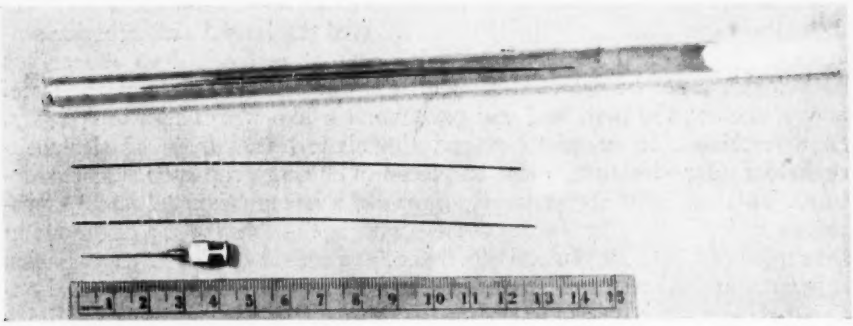

FIG. 1. Electrodes for ventricular electroencephalography.

ventricular needle is in place, the wire is inserted to the proper depth as determined previously by measuring the wire against the needle. The wire is then held steady against a ruler resting on the head and the needle withdrawn over the wire. The wire is held firmly in place with collodion and cotton. The portion of the protruding wire from which the insulation has been removed is wound around a similarly treated length of wire, the other end of which is soldered to a plug-in. These scraped, attached ends are fixed firmly to each other and to a stationary object either with collodion and cotton or with adhesive tape. The wire plug-in is inserted into the plug-in box of a Grass amplifier in the usual manner.

Fig. 1 shows the electrodes marked and prepared for use, with the needle and glass holder. Scalp electrodes are used as well. Since the animals are unanesthetized, they are blindfolded at the time of the recording. Ether anesthesia is used for the ventricular tap, and then several hours are allowed to elapse before the recording is taken. Fig. 2 shows the ventriculogram of a monkey with the wires in place. It will be noted that the wire on the left has slipped out of the ventricle and is lying in brain substance. This happens frequently in monkeys and is apparently due to accidental dislodging of the wire while the needle is being withdrawn over it. For this reason it would probably be preferable to take recordings in humans with the ventricular wire lying undisturbed in the needle. This can be done if the needle is first insulated. As insulating material, we have used cellulose acetate. A needle is dipped into cellulose acetate solution, withdrawn very slowly, and allowed to dry in air. It may then be sterilized in alcohol or by autoclaving.

We have taken a few recordings in anesthetized monkeys with the ventricular wire lying in the insulated needle. An additional advantage of this method of recording is that the needle and the inserted wire may be used as a concentric needle electrode. For this purpose, a small scratch is made in the insulation of the needle close to the bevel, and a wire is attached to

the collar of the needle. A bipolar recording can then be made between the ventricular wire and needle.

Records have been made with a Grass electroencephalograph of ventricular to ground (ear) activity and of ventricular to ventricular activity, and have been compared with simultaneous monopolar recordings of frontal, parietal and occipital cortical regions.

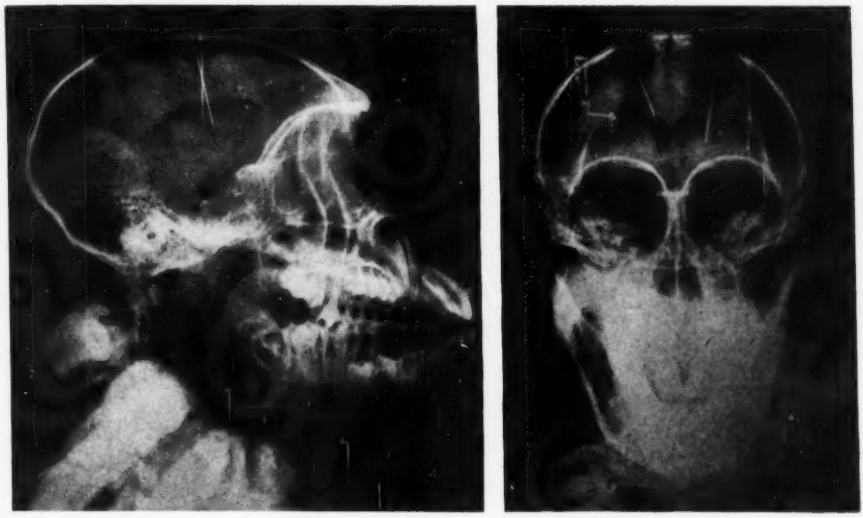

FIG. 2. Ventriculograms showing buried wire electrodes in place. Notice the bilateral, parasagittal bony defect, affording access to the whole length of the lateral ventricles.

RESULTS

Recordings of ventricular electrical activity have been made in three humans* and thirty times in monkeys. Several tentative conclusions may be drawn. The method is safe. No symptoms have been noted while the wires were in place. Several monkeys died of tuberculosis, and postmortem examination of the brain revealed no lesions. The wires have been left in place in the monkeys for six hours without apparent symptoms.

Electrical activity as recorded by ventricular leads usually parallels cortical activity. Under certain conditions, however, recordings of ventricle to ear and of ventricle to ventricle are entirely different from cortical activity (Fig. 3). From this fact we assume that the ventricular leads record the electrical activity of the surrounding subcortical structures and not of the overlying cortex. This assumption is strengthened by the observation that simultaneous tracings from different portions of the ventricle may be completely dissimilar (Fig. 3A). Finally, ventricular leads are of value in lateralizing subcortical lesions. In several monkeys, focal irritative lesions

* These patients were obtained with the cooperation of Dr. Joseph L. Dorsey.

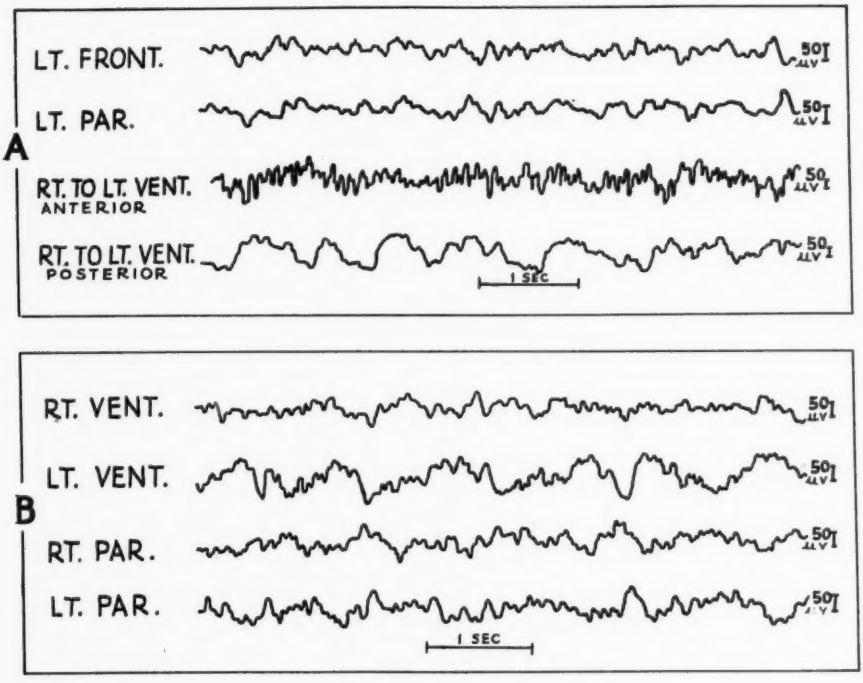

FIG. 3. (A) Simultaneous ventricular and scalp electroencephalogram of an unanesthetized monkey. (B) The same for a monkey with a focal irritative lesion in the neighborhood of the left ventricle.

of various portions of the basal ganglia have produced abnormalities in only one of a pair of symmetrically placed ventricular leads (Fig. 3B).

SUMMARY

A technique is described for recording the electrical activity of the brain by means of wire electrodes inserted in the ventricles. After one and one-half years of experience with monkey recordings, we believe the method to be simple, safe, and adaptable to routine use in conjunction with ventriculography. By application of this method to man, it may be possible to lateralize deep subcortical lesions and to demonstrate the subcortical origin of various abnormal waves.

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SUTURE OF THE POSTERIOR TIBIAL NERVE BELOW THE KNEE, WITH A FOLLOW-UP STUDY OF THE CLINICAL RESULTS

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INTRODUCTION

SUTURE of severed peripheral nerves as early as possible after wounding has been one of the chief objectives in neurosurgery during World War II. This objective has been achieved in the large percentage of peripheral nerve injuries when permissible under the exigencies of war. As a result of early careful nerve suture many cases of excellent regeneration and recovery of function have been followed in the various Neurosurgical Centers in the Zone of the Interior.

One group, however, has been neglected in early nerve suture, and that is the group where the posterior tibial nerve has been severed below the origin of the motor branches to the gastrocnemius and soleus muscles; that is, below the knee.

In cases of nerve paralysis admitted to this hospital, 50 per cent of the patients with paralysis of the peroneal nerve had already had nerve suture, whereas only 7.5 per cent of those admitted with paralysis of the posterior tibial nerve had been recognized previously and had the nerve repaired. There are a number of reasons for this neglect. Many of these cases have been misdiagnosed as partial paralysis because of the presence of normal function of the gastrocnemius and soleus muscles, and in some instances the branches to the flexor hallucis longus and flexor digitorum longus have also been intact. Other cases have been recognized but ignored. In some of the latter anesthesia of the sole of the foot and loss of function of the intrinsic muscles of the foot were noted, but operation on the nerve was felt unnecessary because the patient could plantarflex and invert his foot and plantarflex his toes. In the remainder, suture of the nerve had been considered technically impossible.

Consequently a number of these patients were referred for nerve grafts. In 2 cases in this series it will be noted that nerve graft had been done on previous occasions without success and that ultimately direct nerve suture was accomplished by the method to be described in this report (Table 1). As a result of such errors in diagnosis and faulty reasoning, a considerable number of these patients have been sent back to duty or to convalescent hospitals. Thus much valuable time has been lost from the time of wounding until the patient is finally sent back to a Neurosurgical Center and the nerve suture accomplished.

The importance of carrying out nerve suture in these cases cannot be overemphasized. Normal sensation on the sole of the foot (supplied by the

TABLE 1

Summary of 25 cases of suture of the posterior tibial nerve below the knee

Case No.	Time Between Wounding and Nerve Suture (months)	Preoperative Deficit	Gap Between Nerve Ends		Miscellaneous Remarks
			Before Section (cm.)	After Section (cm.)	
1	12	Anesthesia sole of foot. Loss of intrinsic muscles.	6.5	11.5	At previous operation gap said to be too large. Referred here for nerve graft.
2	6	Anesthesia sole of foot. Loss of intrinsic n uscles.	2.5	7.3	
3	5	Anesthesia sole of foot. Loss of intrinsic muscles.	3.5	8.9	
4	12	Anesthesia of lateral half of ball of foot and undersurface of 3rd, 4th & 5th toes. Incomplete loss of intrinsic muscles of foot.	0	2.2	Lateral neuroma in continuity. Interfascicular dissection & suture of posterior ⅓ of nerve.
5	4	Anesthesia sole of foot. Loss of intrinsic muscles.	3.5	4.5	
6	5	Anesthesia sole of foot. Loss of intrinsic muscles.	1.0	6.0	
7	3	Anesthesia sole of foot. Loss of intrinsic muscles.	1.5	6.5	
8	12	Partial sensory loss of sole. Partial loss of intrinsic muscles.	0	4.5	Neuroma in continuity. Half of nerve sutured.
9	21	Anesthesia sole of foot. Loss of intrinsic muscles. Trophic ulcers on sole of foot.	0	7.0	Neuroma in continuity.
10	18	Anesthesia sole of foot. Loss of intrinsic muscles.	4.3	6.3	
11	5	Anesthesia sole of foot. Loss of intrinsic muscles.	3.0	11.0	
12	6	Anesthesia sole of foot. Loss of intrinsic muscles. Trophic ulcers of sole of foot under heads of 1st & 5th metatarsals.	5.0	9.0	
13	3	Hypesthesia of ball of foot & undersurface of first 3 toes.	0	5.0	Neuroma in continuity. Interfascicular suture of 5 fascicles.
14	5	Anesthesia sole of foot. Loss of intrinsic muscles.	1	5.5	

TABLE 1—*continued*

Case No.	Time Between Wounding and Nerve Suture (months)	Preoperative Deficit	Gap Between Nerve Ends		Miscellaneous Remarks
			Before Section (cm.)	After Section (cm.)	
15	4	Partial anesthesia of sole of foot—lateral half.	0	4.5	Neuroma in continuity. Interfascicular dissection & suture of half of nerve.
16	8	Anesthesia sole of foot. Loss of intrinsic muscles.	2.0	8.0	
17	6	Anesthesia sole of foot. Loss of intrinsic muscles.	3.5	8.5	
18	6	Anesthesia of sole of foot. Loss of intrinsic muscles. Loss of flexor digitorum communis muscle.	2.0	11.8	Nerve damaged in 2 places in 2 episodes of wounding. Neuroma in continuity in lower. Nerve put subcutaneously and medial to soleus muscle.
19	29	Anesthesia sole of foot. Loss of intrinsic muscles.	0	16.0	Had 2 previous unsuccessful nerve graft operations. Frozen-dried nerve graft removed. Nerve placed subcutaneously and medial to soleus muscle.
20	13	Anesthesia sole of foot. Loss of intrinsic muscles.	1	6.5	
21	8	Anesthesia sole of foot. Loss of intrinsic muscles.	3.5	7.0	
22	25	Anesthesia sole of foot. Loss of intrinsic muscles. Trophic ulcers of sole of foot.	7.0	9.5	One previous nerve graft operation had failed. Frozen-dried homogenous nerve graft removed.
23	4	Anesthesia sole of foot. Loss of intrinsic muscles.	0	5.0	Neuroma in continuity.
24	9	Anesthesia sole of foot. Loss of intrinsic muscles.	0	6.5	Neuroma in continuity.
25	6	Anesthesia sole of foot. Loss of intrinsic muscles.	0	5.5	Neuroma in continuity. Nerve put subcutaneously and medial to soleus muscle.

posterior tibial nerve) is of importance for two reasons. In the first place, trophic ulcers on the sole have been noted in several of these patients who had gone for months without nerve repair. Secondly, the sensation as though "walking on air," of which these patients complain, is definitely a handicap to them. Moreover, the loss of function of the intrinsic muscles supplied

by the posterior tibial nerve is a deficit which is important, since not only is it incapacitating so far as balance and finer movements of the foot are concerned, but the integrity of the intrinsic muscles of the foot is essential in maintaining the stability of the arches and in providing soft-tissue support for the angular prominences of the bones.

From 19 February 1945 until 8 September 1945, neurorrhaphy was performed in this hospital in 435 cases. Of this total there were 25 cases (5.7 per cent) in which nerve suture was done for paralysis of the posterior tibial nerve below the origin of the muscle branches to the gastrocnemius and soleus muscles. The study presented in this paper is based on data collected from these 25 cases. In addition to stressing the importance of carrying out early nerve suture, there is detailed description of the operative technique that has enabled us to overcome large gaps in the nerve which until recently had been regarded as technically unsurmountable. The first nerve sutured by the technique to be described was one with a gap of 8.9 cm., which was made up with ease. It was noted that a gap of 11.5 cm. could be made up without tension. This served as a stimulus to the making up of even larger gaps between the nerve ends. In one case there was an incredibly large gap of 16 cm. (Fig. 6) and in three other instances the gaps were 11 cm. or more in length.

The basic principles of the technique are not original, although several refinements have been made in the experience with this series. In Table 1 the important salient features in each case are summarized. It may be seen that an unusually long period of time elapsed between the date of wounding and the time of final direct nerve suture. The average time interval for the entire series was 8.8 months. In 2 cases the interval was more than two years. However, in both these cases nerve grafts had been attempted previously and had failed. Nevertheless, among the 23 remaining cases there were 5 in which this time interval was one year or over, and only 2 patients were operated upon during the optimum interval of three months after wounding.

PLATE I*

FIG. 1. Exposure of neurovascular bundle (posterior tibial artery, vein, and nerve, together with branches) by retracting gastrocnemius and detached soleus muscle *en masse* laterally.

FIG. 2. Beginning mobilization of posterior tibial nerve below popliteal space. (A) Detached medial origin of soleus muscle retracted laterally. (B) Gastrocnemius muscle. (C) Posterior tibial nerve below popliteal space. (D) Posterior tibial nerve above popliteal space.

FIG. 3. Motor branches of posterior tibial nerve before interfascicular separation. Median head of gastrocnemius muscle here is detached from its origin in order to demonstrate anatomic details more clearly. In the operation, the median head of the gastrocnemius muscle need not be detached. The photograph shows entry of the motor branches into muscles they supply: (1) medial head of gastrocnemius; (2) lateral portion of median head of gastrocnemius; (3) lateral head of gastrocnemius; (4) soleus; (5) popliteus and plantaris; (6) posterior tibial; (7) flexor digitorum longus; and (8) flexor hallucis longus muscle. (9) Main trunk of posterior tibial nerve continuing down into foot.

* We are indebted to the Department of Medical Photography at Halloran General Hospital for kind cooperation in the work herein presented.

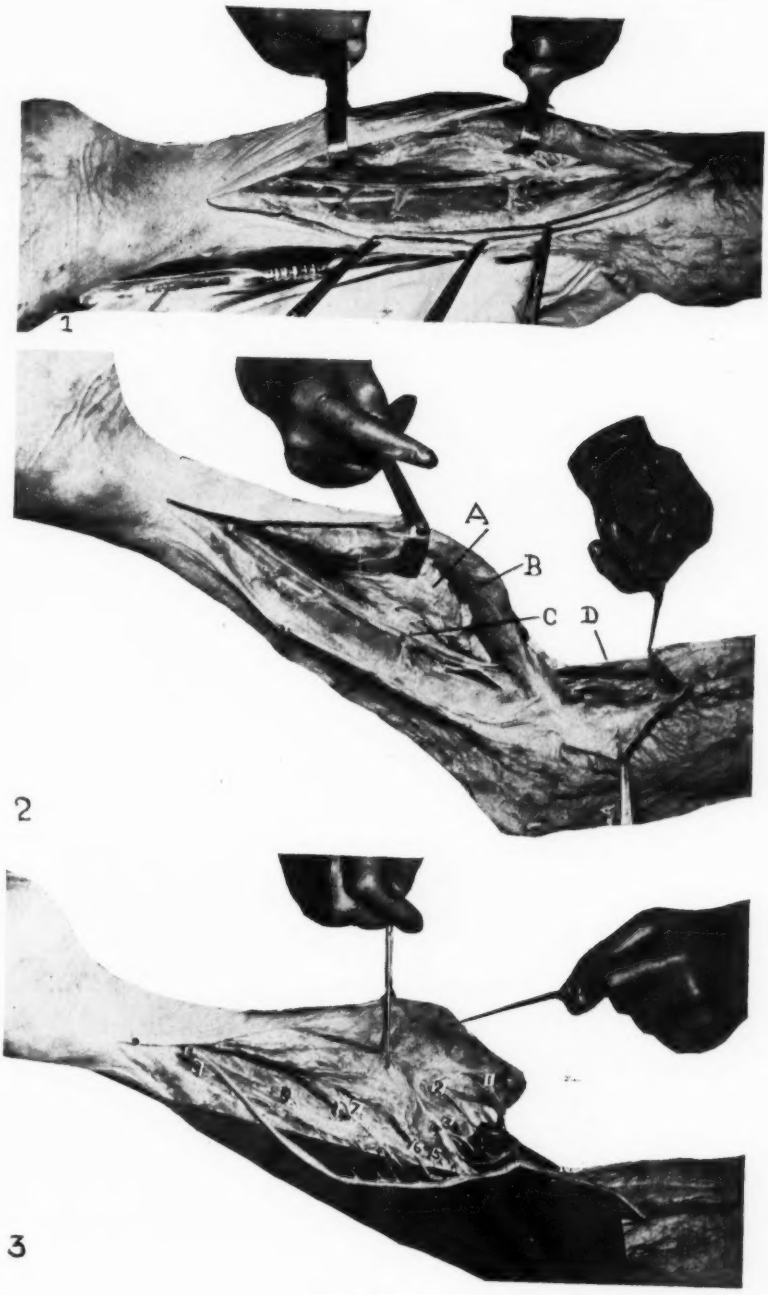

PLATE I

PLATE II

TECHNIQUE

The following detailed description of the operative procedure is representative of the entire group. However, certain modifications and variations are needed when indicated, and will be mentioned later.

The skin incision is begun on the medial aspect of the lower leg, about 2 cm. posterior to the medial edge of the tibia, at the level just proximal to the medial malleolus. The incision extends proximally to the inferior border of the medial condyle of the tibia, then curves laterally and proximally across the flexion crease of the popliteal space, and then upward in the midline of the thigh in its midportion. The incision then is extended downward into the deeper structures and the deep fascia is incised in the direction of the incision. The cleavage plane between the flexor digitorum longus and the posterior tibial muscles anteriorly, and the soleus and the gastrocnemius muscles posteriorly, then is separated in the lower half of the leg. The attachment of the medial border of the soleus muscle is dissected free from the posterior surface of the tibia and from the popliteal line of the tibia. The soleus and the gastrocnemius muscles then are retracted *en masse* laterally. The neurovascular bundle thus is readily exposed as it lies under the deep layer of the fascia cruris on the posterior surface of the posterior tibial muscle, in the groove between the flexor digitorum longus and flexor hallucis longus muscles.

The main trunk of the posterior tibial nerve may be picked up distally with ease at the anteromedial border of the tendon Achilles, just above the internal malleolus as it lies lateral to the posterior tibial vessels enclosed with them in a rather delicate fibrous tissue sheath. Stimulation of the nerve at this point below the site of injury is then carried out to determine whether or not sensory conduction is present.

The nerve then is picked up proximally in the popliteal space, and stimulation at this level is done to determine the integrity of motor conduction to the muscles of the lower leg normally innervated by motor branches of the posterior tibial nerve. The proximal portion of the nerve then is freed distally through the space under the junction of the two heads of the gastrocnemius muscle into the lower portion of the leg, joining the dissection from below.

The proximal neuroma and the distal glioma then are resected and the stumps of the nerve cut back serially to good fascicles. The resulting gap must then be overcome by further dissection in order to accomplish suture of the ends without tension.

By interfascicular dissection, the muscular branches coming off the proximal portion of the posterior tibial nerve then are carefully dissected from the parent nerve as far proximally across the popliteal space into the lower thigh as is necessary to gain enough length to

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PLATE II

FIG. 4. Interfascicular separation of motor branches of posterior tibial nerve has been accomplished proximally to point "X" above the popliteal space. This is the most important step in the procedure. (1) Sciatic nerve. (2) Common peroneal nerve (not in its normal position), which has been mobilized. (3) Posterior tibial nerve in lower part of thigh. (4) Posterior tibial nerve below point to which its motor branches have been mobilized. (5) Common trunk containing branches to the two heads of gastrocnemius, to soleus, and to popliteus muscles. This common trunk has been mobilized to point "X." (6) Branches to posterior tibial muscle and flexor digitorum longus in a common trunk mobilized to point "X." (7) Branch to flexor hallucis longus mobilized to point "X."

FIG. 5. The two severed ends of the posterior tibial nerve here are brought together with the knee flexed. The nerve can be seen to lie subcutaneously in its entire extent and is medial to the median attachment of the soleus muscle; 16 cm. of resected nerve also is shown.

FIG. 6. Specimen from Case 19 in which removal of frozen-dried nerve homograft and resection of proximal and distal stumps created a deficit of 16 cm. in posterior tibial nerve. *Upper:* Cross sections of proximal and distal stumps representative of ends finally sutured, and median longitudinal section of the graft and proximal stump. *Lower:* External view of the graft with separate cables bound together by ingrowing fibrous tissue, and, at extreme right, median longitudinal section of distal stump.

TABLE 2

Follow-up study of clinical results

Case No.	Time from Operation to Last Examination (months)	Advance of Tinel's Sign (cm.)	Sensory Evaluation* (graded 0–4)	Skin Resistance† (graded 0–2)	Clinical Appraisal
1	6	40	1	1	Good sensory return to sole of foot.
2	6	35	1	1	Good sensory return to sole of foot.
3	6	55	1	1	Good sensory return to sole of foot.
4	6	40	2	2	Nearly complete sensory return. Evaluation difficult because only $\frac{1}{3}$ of nerve sutured.
5	9	45	1	1	Beginning sensory return; patient able to judge when foot is on ground.
6	9	25 Practically complete distal advance.	2	1	Good sensory return.
7	9	Complete distal advance.	2	1	Excellent sensory return.
8	6	50	2	1	Excellent sensory return.
9	9	Complete distal advance.	2	1	Excellent sensory return.
10	3	15	0	0	Too early for sensory return to appear.
11	6	only 10	0	0	X-ray suggests separation of nerve ends at suture site.
12	9	Tinel's completely advanced.	2	1	Excellent sensory return in sole of foot.
13	9	Tinel's 45 cm. (practically completely advanced).	2	1	Very good sensory return in sole of foot.
14	10	35 (practically complete).	1	1	Good sensory return.
15	6	35 (practically complete).	1	1	Good sensory return.

TABLE 2—*continued*

Case No.	Time from Operation to Last Examination (months)	Advance of Tinel's Sign (cm.)	Sensory Evaluation* (graded 0–4)	Skin Resistance† (graded 0–2)	Clinical Appraisal
16	6	45	1	1	Good sensory return.
17	6	40	1	1	Deep cutaneous pain in sole of foot.
18	9	35 (Nearly complete)	2	1	Good sensory return.
19	6	20	0	0	No sensory return. X-ray reveals intact suture site. Leg is completely straightened.
20	6	40	1	1	Good sensory return.
21	6	35	1	1	Good sensory return.
22	3	20	0	0	No sensory return yet. X-ray reveals intact suture site. Too early to expect return.
23	3	22	0	0	Too early for sensory return.
24	3	20	0	0	Too early for sensory return.
25	3	10	0	0	Too early for return. X-ray reveals suture site to be intact.

* Sensory Evaluation (usual modalities):
0—absence of sensibility in autonomous zone.
1—recovery of deep cutaneous pain in autonomous zone.
2—recovery of some degree of superficial pain and touch in autonomous zone.
3—return of pain and touch in autonomous zone without over-response.
4—recovery as in 3 with two-point discrimination in autonomous zone.

† Skin Resistance:
0—persistent area of increased skin resistance.
1—diminution of area of increased skin resistance.
2—normal skin resistance.

make up the gap between the cut ends of the nerve. After mobilization of the proximal portion of the nerve together with its muscular branches, stimulation of the nerve proximally reveals the presence of normal motor conduction of the muscular branches that have been freed from the parent nerve. The parent posterior tibial nerve is pulled down and, with the knee flexed to 90 degrees, the ends may be brought together without tension.

The ends of the nerve are sutured together with interrupted .003 tantalum sutures in the epineurium, and a narrow cuff of tempered tantalum foil, 1.0 cm. in length, is placed about the anastomotic site.

The attachment of the soleus muscle then is sutured back to the popliteal line of the tibia

with a few interrupted 0000 silk sutures, and the wound closed in layers with interrupted 0000 silk sutures in the deep fascia, subcutaneous tissue and skin.

A firm pressure dressing is applied, using an Ace bandage, and the knee is maintained at 90 degrees flexion by means of a plaster of Paris double hip spica.

Variations and modifications of the above-mentioned technique of the operation have been used where indications have arisen. In some cases a different skin incision was made in the region of the popliteal space. Thus, instead of crossing the flexion crease in the popliteal space at an angle, the incision was extended proximally and continued up the medial aspect of the popliteal space, thus avoiding the flexion crease entirely. In such cases the skin flap was undercut to the popliteal fascia and the flap reflected laterally. This variation has proven entirely satisfactory. However, in none of the cases where the line of incision crossed the flexion crease was there any web contracture of the skin. Of course, care was taken to curve the incision in the line of the flexion crease and not to cross it at a right angle.

In some cases where the gap between the cut ends of the nerve was considerable (Case 19), it was necessary to pull the proximal parent nerve upward through the space under the two heads of the gastrocnemius muscle and place it subcutaneously across the popliteal space and downward along the medial border of the soleus muscle. In such cases also it has been necessary to free the muscular branches from the parent nerve upwards into the midthigh and even further proximally. In this case the sciatic nerve also was freed up to the level of the lower edge of the gluteus maximus muscle. In some cases the common peroneal nerve then was likewise separated from the sciatic nerve in order to aid in mobilizing the posterior tibial nerve.

For the purpose of illustrating the technique in mobilizing the posterior tibial nerve in order to overcome a large gap, dissections on the cadaver were done (Figs. 1–5). Thus it can be seen that 16 cm. of the nerve can be removed, and the intervening gap made up by the mobilization and the remaining cut ends of the nerve brought together. This essentially was the technique used in Case 19, in which there was a final gap of 16 cm. (see Fig. 6 for the specimen removed in this case).

The essential steps in the procedure are indicated in the series of photographs. Fig. 3 is included to illustrate the motor branches that come off the parent nerve below the popliteal space and the muscles that they innervate. Fig. 4 shows these same branches freed from the parent nerve to a point above the popliteal space by interfascicular dissection. This is the most important step in the operation in order to gain length in the nerve and still preserve the motor conduction of these branches.

The other details are given in the legends accompanying the various photographs.

FOLLOW-UP STUDY OF CASES REPORTED

Follow-up studies on all nerve sutures done are made at intervals of three months after operation. Enough time has now elapsed following nerve suture in the cases herein reported to appraise their clinical results. These have been tabulated in Table 2. Thus it will be noted that all cases show satisfactory progress with one exception (Case 11). Roentgenologic examination in that case revealed evidence of separation of the suture site. The apparent distal advance of 10 cm. of Tinel's sign, with no subsequent distal advance, might be explained on the basis of transmission from the proximal end of the separated nerve.

The remaining cases are gratifying in their results. All show definite sensory return except 7, and these have not been observed long enough for return to be possible. Some of the patients have been returned to civilian life and it is felt that their improvement will continue. The questions as to whether the function of the intrinsic muscles of the foot will return to nor-

mal and whether there will be achievement of two-point discrimination in sensory recovery, are unanswered at the time of this writing. Doubtless, a considerable further period of time must elapse before these questions will be solved.

SUMMARY AND CONCLUSIONS

1. A series of 25 cases of neurorrhaphy of the posterior tibial nerve below the popliteal space is presented.

2. The importance of suture of the posterior tibial nerve in cases where it has been severed below the popliteal space, and in which the chief deficit is loss of sensation of the sole and of the function of the intrinsic muscles of the foot, is emphasized.

3. In one case in this series there was a gap of 16 cm. between the ends of the sectioned nerve, and in 3 other cases gaps of 11.0 cm. or more were overcome.

4. Cadaver dissection to illustrate the technique by which large gaps between the sectioned ends of the nerve can be overcome was carried out, and photographs of the dissection are included.

5. Postoperative follow-up studies at intervals of three months reveal satisfactory sensory return.

REVERSIBILITY OF CEREBRAL VENTRICULAR DILATATION

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VENTRICULOGRAPHY and encephalography, combined or separate, are established procedures in the preoperative diagnosis and localization of mass lesions of the brain, but seldom have they been utilized to study the evolution of anatomic changes following operation. In the course of a larger study being carried on at the Hospital of the University of Pennsylvania, we have had occasion to observe, by ventriculography and ventriculo-encephalography, three patients with mass lesions causing an internal hydrocephalus, before and after relief of the hydrocephalus. The significant feature that we wish to emphasize in this report is that dilatation of the lateral ventricles caused by obstructing lesions in the third or fourth ventricle may, in certain instances, be reversible.

REPORT OF CASES

Case 1. O.B., an 18-year-old man, complained of headache and blurring of vision for 3 months and staggering gait for 3 weeks before entering the hospital. On examination, generalized hypotonia and hyporeflexia were evident and there was marked trunkal ataxia. His optic discs were elevated to between 5 and 6 D. The preoperative ventriculographic study is shown in Fig. 1, A and B.

At operation on Aug. 15, 1943, most of a large medulloblastoma of the vermis of the cerebellum was removed. This immediately relieved the obstruction of the fourth ventricle. The postoperative convalescence was uneventful. In January 1944 a full course of roentgen therapy was administered.

Although the patient continued to do well, a second course of roentgen therapy was planned. A second ventriculo-encephalographic study was done on Sept. 19, 1944, 13 months after his original air study (Fig. 1, C and D). The lateral and third ventricles were approximately normal in size. One can compare the appearances before and after operation because the same roentgen technic was employed in both examinations. A filling defect was noted in the fourth ventricle which was thought to be residual neoplasm.

In May 1945 a recurrence of symptoms was noted and the patient died on Oct. 2, 1945.

Case 2. M.W., a 38-year-old woman, had headache and blurring of vision beginning in December 1943. Nausea, vomiting and increasing somnolence began in January 1944. On admission to the University Hospital April 11, 1944, she was disoriented and confused, unable to stand or walk and had a high degree of papilledema bilaterally.

A ventriculographic study (Fig. 2, A and B) was performed on April 12, 1944, and immediately followed by a suboccipital craniectomy. A fairly well demarcated medulloblastoma was removed from the vermis of the cerebellum. The obstruction of the iter was relieved and cerebrospinal fluid was seen to flow freely from it. Her convalescence was uneventful and on July 7, 1944, 3 months after the initial air study, a ventriculo-encephalogram was done preceding irradiation therapy. This showed considerable decrease in the size of the ventricles (Fig. 2, C and D).

In November 1944, she noted pain low in the back, radiating down both legs. Otherwise, save for poor vision and mild residual disturbance in walking, she was asymptomatic. An-

FIG. 1. *Case 1.* (A and B) Ventriculogram of an 18-year-old man who had symptoms of a posterior fossa lesion for 3 months. In the postero-anterior and left lateral horizontal positions, the lateral and 3rd ventricles are dilated to approximately twice their normal volume. The foramina of Monro and the aqueduct of Sylvius also are dilated. The shadow of the 4th ventricle is not visible. (C and D) Ventriculoencephalogram 13 months after operative removal of a cerebellar medulloblastoma, showing little dilatation of the lateral and 3rd ventricles.

other course of x-ray therapy was given in December 1944, which included irradiation over the vertebral column, without improvement of her low back pain. She was again readmitted in March 1945, because of back pain. Spinal fluid manometric studies were entirely normal as was the spinal fluid total protein. Pantopaque myelography was entirely negative. She was discharged, and a firm brace was recommended for her back. She was last seen on June 25, 1945 and her back pain was much relieved.

Case 3. H.R., an 8-year-old boy, was struck on the head 2 months before admission to the University Hospital. He was not unconscious but did complain of generalized headache immediately after the injury. He had occasional headaches for the following month, which subsequently increased in frequency to the point where he complained daily of headache in the morning on awakening. Nausea and vomiting accompanied each bout of morning headache. On admission he had 5 D. of papilledema bilaterally, accompanied by hemorrhages in the left eye. The visual fields were normal but his visual acuity was reduced in each eye to

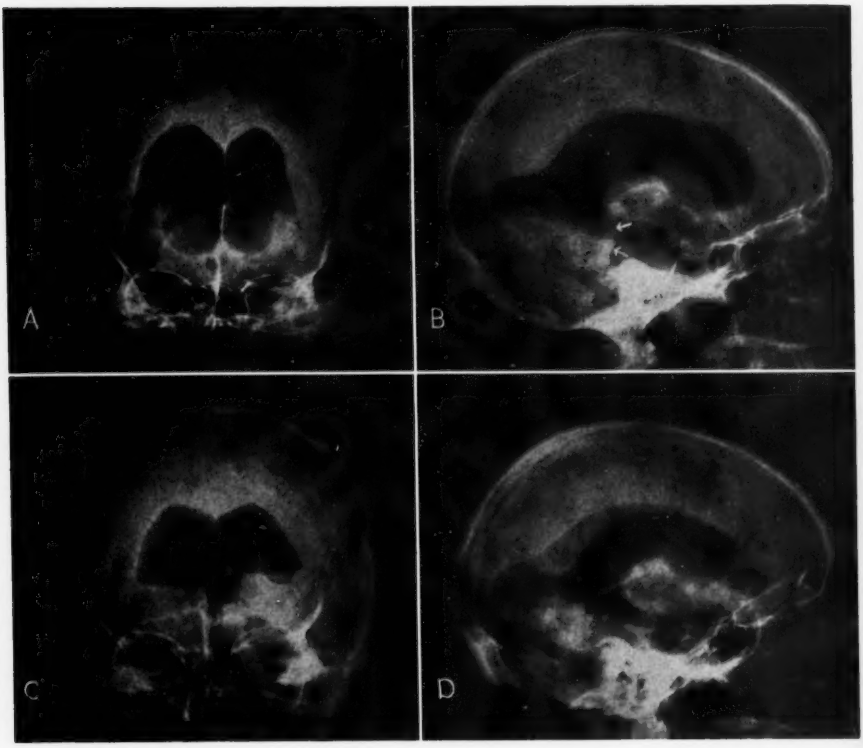

FIG. 2. *Case 2.* (A and B) Preoperative ventriculogram of a 38-year-old woman with symptoms of increased intracranial pressure for 4½ months. The postero-anterior and left lateral horizontal views reveal lateral ventricles enlarged approximately 3 times. The aqueduct of Sylvius and 4th ventricle are displaced forward by a mass in the region of the cerebellum (arrows). (C and D) Ventriculo-encephalogram made 3 months after operative removal of a cerebellar medulloblastoma. The lateral ventricles are approximately ½ their former size. The absence of the shadows of the subarachnoid pathways may be secondary to basal arachnoiditis, residual tumor, or failure of complete drainage of the cerebrospinal fluid.

6/15. The procedure of ventriculo-encephalography, done on June 19, 1945, showed the lateral ventricles to be symmetrically dilated, due apparently to a mass in the anterior portion of the third ventricle obstructing the foramina of Monro. The posterior portion of the third ventricle and fourth ventricle appeared normal (Fig. 3, A and B). A right frontotemporal craniotomy with craniectomy was done for decompression and to allow the right lateral ventricle to dilate sufficiently to permit an operative attack on the presumed third ventricle tumor at a later time. The child did well postoperatively and was discharged asymptomatic on June 29, 1945.

He was readmitted to the hospital on Sept. 15, 1945. In the interim he had been entirely free of all symptoms of increased intracranial pressure. He had, however, gained an excessive amount of weight. The papilledema had receded, leaving an unusual amount of consecutive atrophy of the optic papillae. His acuity was maintained at 6/15 bilaterally, but the visual fields showed a left homonymous superior quadrantic defect. Another ventriculographic study was done on Oct. 2, 1945 (Fig. 3, C and D). The lateral ventricles were almost normal

FIG. 3. *Case 3.* (A and B) Preoperative ventriculo-encephalogram of an 8-year-old boy with symptoms of increased intracranial pressure for 2 months. There is moderate dilatation of the lateral ventricles and a filling defect is in the third ventricle. (C and D) Ventriculogram 3½ months after operation shows that the lateral ventricles are much smaller and the 3rd ventricle deformity is not so apparent.

in size. The third ventricle was much more completely filled with air. Since the child was relatively asymptomatic, it was felt that operation could be deferred. The child was discharged to return for further observation in 3 months. However, he had an acute recurrence of the symptoms of increased intracranial pressure, and on Dec. 8, 1945 reoperation disclosed a cystic tumor of the third ventricle.

COMMENT

In the first patient (Fig. 1) there was an internal hydrocephalus, secondary to a tumor in the region of the fourth ventricle, which almost disappeared

after operation. The patient had had symptoms of increased intracranial pressure for 3 months and at the time of the ventriculogram the lateral ventricles, the foramina of Monro and the third ventricle were at least twice their normal size. Approximately 14 months after operation, ventriculo-encephalograms showed essentially normal ventricles. In the second patient (Fig. 2) there was greater dilatation of the lateral ventricles than was observed in the first patient. Three months after operation there was some reduction in the size of the ventricles, but they were still larger than normal. In this instance, the greater initial dilatation of the ventricles, the shorter interim between studies, the longer duration of symptoms and the difference in the ages of the patients may be the explanation for the incomplete restoration to normalcy of the ventricular system. In the third patient (Fig. 3) the enlargement of the lateral ventricles was due to an obstructing lesion of the third ventricle. Following decompression the obstruction was relieved temporarily and in 3½ months the lateral ventricles were considerably smaller.

Penfield[2] states that a temporary internal hydrocephalus may leave the ventricles dilated for the remainder of the patient's life. He suggests that it is the result of "an atrophy which, to a large extent, remains permanent." Northfield,[1] in a recent discussion of this point, concludes it is not known whether ventricles remain permanently dilated after transient increased intracranial pressure. The cases we are reporting effectively demonstrate that ventricular dilatation can be a reversible phenomenon.

The extent of the reversible changes are different in all of the three patients. Our experience is too limited to permit drawing any conclusions, but increased use of ventriculo-encephalograms before and after operation, as well as before and after roentgen therapy, should provide data from which one may learn more about dilatation of the ventricles due to obstructing lesions, and how reversible those changes are after the relief of the obstruction at varying intervals.

SUMMARY

Three cases are reported of internal hydrocephalus caused by an obstructing lesion in either the third or fourth ventricle which were studied by ventriculography before and ventriculo-encephalography after relief of the obstruction. In every patient, the lateral ventricles were smaller after operation, illustrating, we believe, that ventricular size is reversible in many instances, if the dilatation of the ventricles is due to an obstructive lesion, and the obstruction has not been present too long.

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PENETRATING CRANIOCEREBRAL INJURIES

EVALUATION OF THE LATE RESULTS IN A GROUP OF 200 CONSECUTIVE PENETRATING CRANIAL WAR WOUNDS

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A GROUP of 200 consecutive cases of severe craniocerebral trauma due to combat injury has been studied in an effort to evaluate the status of the patients at the time of disposition from an Army General Hospital. There have been excellent reports on the early treatment and its results in this type of injury.[2,3,4,7,8,11,15] Many of the cases in this report have been discussed in their more acute phases in such papers as those of Schwartz and Roulhac,[15] Haynes,[11] and Campbell.[4] However, there have been no attempts to evaluate the results at a later stage. It is obvious, from the time that has elapsed following the injuries, that the present report does not constitute a study of the end results. Many patients will continue to show improvement at home or in the various Veterans Facilities to which they have been sent, and unfortunately not a few of them will become worse. This may be especially true in respect to the development of convulsive disorders. This study, therefore, might be considered as being done in an intermediate or "subacute" stage of this type of injury. Though later studies naturally have not been done on soldiers injured in the second world war, more "chronic" studies have been made in a similar group of injuries in the first world war by Ascroft,[1] Cushing,[6] Wagstaffe and Adie,[17] Wagstaffe,[16] and Goldstein.[10]

The basic criterion in the selection of the present group of patients was dural penetration. All the injuries were incurred in combat. Slightly over 90 per cent of this group were injured in either the European or Mediterranean theaters of operation. The remaining 10 per cent were wounded in the South Pacific theater. The original injuries were sustained between August 1943 and April 1945. Table 1 gives the type of missile or projectile causing the injury. The great majority of injuries were due to artillery shell fragments. In this series at least, other types of weapons caused relatively few severe penetrating wounds of the head.

There seemed to be little remarkable about the location of the injury. Table 2 summarizes the various sites. Many of these injuries included more than one of the usual areas of the brain, but it seemed most satisfactory to report them by judging the approximate area of greatest brain damage as well as it could be determined from the location of the skull defect and from

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a review of the original operative report describing the early debridement. There were 106 left-sided wounds of entrance and 90 on the right side. It might possibly be of interest and value in the future design and reinforcement of protective headgear to mention that 36 per cent or over $\frac{1}{3}$ of the

TABLE 1

Missiles causing penetrating wounds of head

Artillery shell fragment	172	86.0%
Rifle bullets	12	6.0%
Machine gun bullets	5	2.5%
Small arms	4	2.0%
Anti-aircraft shell fragments	3	1.5%
Aerial bomb fragments	2	1.0%
Land mine fragments	1	.5%
Airplane crash	1	.5%
TOTAL	200	100 %

injuries were primarily in the parietal regions of the skull. One patient in this group sustained two different penetrating head wounds while another received three separate injuries.

In reviewing this group of injuries there were several outstanding facts

TABLE 2

Location of injury

Right frontal	24
Left frontal	27
Right temporal	19
Left temporal	14
Right parietal	29
Left parietal	44
Right occipital	15
Left occipital	18
Midoccipital	4
Right eye	3
Left eye	3
Vertex	2
Midfrontal	1
TOTAL	203

that seemed to warrant more detailed study. These were concerned particularly with the incidence of infection, the nature and frequency of convulsive disorders, the presence or absence of unconsciousness and its duration, and finally the ultimate disposition of patients, with mention of the methods used in therapy and rehabilitation.

INFECTION, DEBRIDEMENT AND EARLY TREATMENT

There were 47 cases (23 per cent) in which there was definite evidence of infection. In considering the problem of infection it is extremely important to try to evaluate the original treatment, especially as regards the time and

extent of debridement and the use of drug therapy. The technique of early treatment and the use of chemotherapeutic agents have been well covered in the reports of Campbell,[4] Schwartz and Roulhac[15] and Haynes.[11] Moreover, Rowe and Turner[14] have emphasized some of the important differences and peculiarities encountered in war wound infections of the brain. Table 3 presents some of the facts about the debridement, the presence of foreign bodies, and the incidence of epilepsy in this group of 47 patients with infections.

In the entire group of 200 injuries there was a definite statement in the record in 178 instances that gave some information about the type and time of original debridement. This time interval varied from 2 hours to 3 weeks or 504 hours. The average time of debridement following the injury was 33 hours. However, in only 31 instances (17 per cent) was the debridement over 24 hours after the injury, but in 10 of these it was well over 100 hours.

There were 8 patients in whom either no or inadequate intracranial debridement was carried out in spite of dural penetration. Five had orbital or periorbital penetrations, and none of these developed infection. The other 3 had only superficial scalp debridements because penetration was not suspected. Of these, one had debridement in a German Hospital and complete debridement was carried out by Allied medical officers four days later. The second was debrided two months later, bone fragments being removed because of the complaint of headache. The third patient never had more than scalp debridement. These three never showed any signs of infection.

Of the 47 cases with infection, 15 could be considered as superficial wound infections and were inconsequential in increasing the time of convalescence of the patient. On the other hand, there were 32 instances of what should be regarded as deep or major infections. These included 17 brain abscesses. Two of these were complicated by meningitis, one by a subdural abscess, and one was followed by a persistent cerebral fungus. In one case of meningitis with recovery, the presence of B. Welchii infection of the cerebral cortex was proven bacteriologically. There were 6 instances in which there was persistent leakage of cerebrospinal fluid from the wound. There were 6 cerebral fungi that developed following wound infections. In 3 patients meningitis occurred after the original debridement.

In 45 of the 47 patients with infection the time of the original debridement was available. The average time was 25.3 hours after injury, which is definitely shorter than that for the entire group. In no instance of infection was the time of debridement over 72 hours. Table 3 indicates that the time of debridement following injury is essentially the same in both superficially and more extensively infected cases. These statistics do not entirely agree with those given by Haynes.[11]

All patients in the entire series of 200 were given some type of chemotherapy during their early treatment. The majority had sulfanilamide applied locally in the forward areas, and then were placed on sulfadiazine orally or parenterally following the debridement. About ¾ of the patients received

TABLE 3

Infection

No.	Type	Debrided (Hrs. after Injury)	Epilepsy	Foreign Bodies*
1	Superficial (W)	24	No	M
3	Superficial (W)	15	No	N
4	C.S.F. leak	36	No	N
8	Superficial	?	No	N
9	Superficial	10	No	N
11	Brain abscess, meningitis	10	Yes	N
13	Fungus	?	Yes	M
14	Brain abscess	6	Yes	N
19	Superficial	27	No	N
24	Superficial	24	Yes	M
26	Fungus	7	Yes	N
27	Superficial	20	No	N
36	Brain abscess	17	Yes	M
37	Fungus	24	No	M/B
43	Fungus	24	No	N
44	Brain abscess	30	No	N
45	Brain abscess	24	Yes	N
52	Superficial	24	No	N
57	Superficial	10	No	N
64	C.S.F. leak	24	No	M
81	Superficial	72	No	N
101	Brain abscess, subdural abscess	24	Yes	N
108	Meningitis	72	No	M/B
113	Brain abscess	24	No	N
118	C.S.F. leak	24	No	M
119	Fungus	?	No	M/B
130	Superficial	48	No	B
135	C.S.F. leak	24	Yes	M
139	Fungus	24	No	N
140	C.S.F. leak	24	Yes	M
149	Brain abscess, fungus, meningitis	26	No	N
150	Brain abscess, fungus, multiple, gas infection?	24	Yes	N
152	Meningitis	24	No	N
153	Brain abscess	12	No	M/B
158	Brain abscess, fungus	24	No	M
162	Brain abscess	24	Yes	M
164	C.S.F. leak	48	No	N
170	Superficial	48	Yes	B
173	Brain abscess	16	No	N
178	Brain abscess	24	No	M/B
184	Meningitis	18	No	N
189	Brain abscess	24	No	N
190	Brain abscess	48	Yes	N
195	Brain abscess	16	No	M
196	Superficial	3	No	N
197	Superficial	48	No	M
200	Rhinorrhea, superficial	10	Yes	M/B

* N—None M—Metal B—Bone M/B—Metal and Bone.

penicillin in large doses during their early stages of treatment. As far as could be determined from the overseas records, the drug therapy in the 47 cases showing infection was essentially the same as that for the whole group.

Foreign bodies retained in the brain substance seemed to have little influence on the presence of infection. Of the 47 patients with infection, 21 (44.6 per cent) showed retained fragments, of which 13 were metallic, 6 both bone and metallic, and 2 bone only. In the remaining 153 men without infection, x-rays revealed retained matter in 73 (47 per cent), of which 56 were metallic, 8 mixed metal and bone, and 9 bone only.

The study of the cases in which infection followed the original debridement confirms the opinion of Cushing,[5] Horrax[12] and many others that the interval prior to debridement, the presence of foreign bodies, and the use of drug therapy are important but are not the cardinal factors in the prevention of infection in the treatment of penetrating wounds of the brain. The findings in this series again demonstrate the almost universal opinion that the most important factor is careful and complete debridement with adequate facilities and personnel to accomplish a neurosurgically sound cerebral cleansing.

EPILEPSY

The incidence and nature of convulsive disorders following this type of injury is of primary importance. Goldstein,[10] Ascroft[1] and Wagstaffe,[16] in studying patients injured in the last war, have reported the frequency of epilepsy in those soldiers having dural penetration as varying between 40 and 81 per cent. The reports were made for the most part several years after the original injury. Undoubtedly there will be more patients in the present series who will develop convulsive seizures.

There are 34 (17 per cent) patients who have had some kind of convulsive seizure since their injury, as shown in Table 4. One of these was a known epileptic since childhood and his seizures have continued unchanged since he was wounded. His case should therefore be excluded here. There were only 2 instances of minor or "petit mal" epilepsy, and one other patient had had both major and minor convulsive episodes. It is interesting that in only 4 soldiers was it possible to observe or obtain any history of a Jacksonian march. This seems unusual considering the localized character of many of the lesions.

One-third of the patients with a history of epilepsy had from one to two grand mal attacks shortly after their injury or within a few days of the acute treatment. Only 3 continued to have epilepsy at the time they were on the neurosurgical section of Ashford General Hospital. The remaining $\frac{2}{3}$ of the patients developed seizures from 2 weeks to 8 months after the acute treatment had been completed, and the majority of these continued to have spells until controlled medically. It may be reasonable to assume that the prognosis for the future in the first group is more favorable, but it is felt that even here it must be extremely guarded.

TABLE 4

Epilepsy

No.	Debrided (Hrs. after Injury)	Infection*	Length of Unconsciousness	Foreign Body†	Location	Type‡
2	30	No	3 min.	Yes (M)	R.P.	G.M.
7	6	No	?	No	L.F.	G.M.
10	?	No	24 hours	No	L.T.	G.M.
11	10	Yes (D)	None	No	R.F.	G.M.
12	?	No	48 hours	No	L.F.	G.M.
13	?	Yes (D)	?	Yes (M)	L.T.	G.M.
14	6	Yes (D)	None	No	R.T.	G.M.
15	8	No	8 days	Yes (B)	L.F.	G.M.
16	12	No	?	No	L.F.	G.M.
21	12	Yes (S)	14 days	Yes (M)	R.F.	J.G.M.
24	24	Yes (D)	14 days	No	R.P.	G.M.
26	7	Yes (D)	24 hours	No	L.F.	G.M.
36	17	No	1 min.	No	L.P.	P.M.
45	24	Yes (D)	2 days	Yes (M)	L.F.	G.M.
62	8	No	3 min.	No	L.T.	G.M./P.M.
66	168	No	10 min.	Yes (M/B)	R.P.	G.M.
82	4	No	?	No	R.P.	G.M.
101	24	Yes (D)	21 days	No	R.F.	J.G.M.
105	72	No	None	No	M.F.	G.M.
116	20	No	?	No	R.P.	G.M.
133	18	No	?	Yes (M)	R.P.	G.M.
135	24	Yes (D)	?	Yes (M)	L.F.	G.M.
138	24	No	10 days	No	R.O.	G.M.
140	24	Yes (D)	?	Yes (M)	R.F.	P.M.
141	24	No	2 days	No	R.P.	G.M.
150	24	Yes (D)	?	No	L.P.	G.M.
154	10	No	?	Yes (B/M)	R.F.	G.M.
155	24	No	10 days	Yes (M)	L.F.	G.M.
159	24	No	None	Yes (M)	L.P.	G.M.
162	24	Yes (D)	None	Yes (M)	R.P.	J.G.M.
170	48	Yes (S)	?	Yes (B)	M.O.	J.G.M.
175	11	No	45 min.	No	V.	G.M.
190	48	Yes (D)	30 min.	No	R.F.	G.M.
200	10	Yes (S)	48 hours	Yes (M/B)	L.F.	G.M.

* D—Deep infection S—Superficial infection
† M—Metal B—Bone M/B—Metal and Bone
‡ G.M.—Grand mal J.G.M.—Jacksonian grand mal P.M.—Petit mal

Of the 34 patients with convulsive seizures, 19 (56 per cent) showed no evidence of wound infection at any time. Of the 15 remaining patients, 12 had major or deep infection, that is, brain abscess, meningitis, cerebral fungus and cerebrospinal fluid leaks. It seems probable that the superficial wound infection in the other 3 had little to do with the onset of convulsive seizures. In all but 3 of these cases the time of debridement was available. The average time was 25 hours or less than that for the whole series and essentially the same as in the group of 47 patients with infections.

In the records of 23 of the 34 soldiers with convulsive seizures, some statement as to the presence of unconsciousness and its duration was available. In 5 cases there was no period of unconsciousness following the injury while in the remaining 18 it varied from 3 minutes to 21 days.

Nineteen (56 per cent) of the patients showed no x-ray evidence of retained foreign bodies. There were 10 with residual metallic foreign bodies in the brain parenchyma as judged by stereo x-rays, 3 with both metal and bone remaining, and 2 with bone fragments only. These figures do not show any statistically valid variation from the incidence of foreign bodies in the patients who had never developed convulsive seizures. This tends to confirm the opinion of Penfield[13] and others that it is usually the cerebrodural scar that is epileptogenic and not necessarily the foreign body or the foreign body reaction.

It is doubtful if this group gives any valid information about the incidence of convulsions in relation to the various areas of brain injured. Frontoparietal injuries predominate in the group with convulsions, but this probably only reflects the fact that 66 per cent of the total series of injuries were in this region.

There were 14 patients in this group in whom one or more electroencephalographic tracings were taken. Seven, or $\frac{1}{2}$ of these, showed no focal signs and only diffuse abnormality. In one of the 7 patients reported as having focal abnormality, the only evidence of this was increased amplitude in the region of the cranial defect. The remaining 6 patients showed definite localized abnormalities in form, frequency and amplitude. However, 5 of these 6 also were reported as having concomitant diffuse abnormal changes in their electroencephalograms. In comparing the electroencephalographic tracings of this group with clinical convulsive seizures with the records of patients with dural penetration but without clinical epilepsy, there seemed to be little variation. One, however, is impressed with the low incidence of focal EEG changes, and the frequency with which diffuse changes accompany local changes when present. These facts plus the infrequent finding of Jacksonian epilepsy strongly suggest that early attempts at local cortical scar resections, either prophylactically or therapeutically, may be of little value. However, this group is too small to come to any definite conclusions. It has been possible to control the convulsions in every patient by a carefully regulated dosage of dilantin and/or phenobarbital.

RESIDUA

Table 5 is a tabulation of the various types of neurological signs and symptoms found as residua in this group of soldiers. These findings are naturally only a result of the location and severity of the brain damage.

Forty patients (20 per cent) had no residua other than the cranial defect, and there were 15 more whose only residuum was convulsive seizures. Hemiparesis of all degrees, from slight residual weakness to complete spastic hemiplegia, was by far the most common neurological defect. All had shown

some improvement as compared with the description of their original condition following injury, but in many instances this had been minimal. Dysphasia and its allied disorders was about one-half as frequent as motor weakness, while visual field changes followed closely. It is interesting to note the relative paucity of headaches and dizziness in this group of severe injuries, symptoms which are so extremely common in any large group of closed head injuries, either in civilian or military practice.[9]

In 150 instances, or 75 per cent of the total series, some statement could be found as to the presence or absence and duration of unconsciousness following the acute injury. In 52 (34.7 per cent) records there was a definite statement that at no time was there any loss of consciousness. The periods of loss of consciousness were described as varying from momentary to 6

TABLE 5

Residua: neurological symptoms and signs

Hemiparesis	66
No residua	40
Headache	35
Dysphasia and allied disorder	33
Visual field defects	29
Dizziness and vertigo	19
Convulsive seizures only residuum	15
Defective hearing	15
Mental deterioration, personality changes	14
Facial paralysis	8
Sensory deficit (marked)	6
Blindness	3
Olfactory disturbance	3
Tinnitus	3
Diplopia	2
Fifth nerve changes	1
Ataxia	1

weeks. In 23 (15 per cent) instances the period of unconsciousness was less than 5 minutes.

DISPOSITION

Definite evaluation of the ultimate disposition in this group is difficult because many have been injured so recently. Many of these men will show improvement for as long as 2 years after leaving Ashford General Hospital. It is doubtful if any will regress in respect to their neurological signs, but, as stressed in the section on epilepsy, some undoubtedly will develop convulsive seizures as time goes by.

Table 6 gives the disposition in this group of 200 patients with dural penetration. There were 174 (85 per cent) separated from the service either by certificate of disability or retirement. This figure may seem large, but it is influenced by the military necessity and policies of the Medical Department, as well as by the actual physical condition of the individual patients. Of these, 144 (82 per cent) could be returned to their homes, and were

considered capable of being completely or partially self-supporting. Twenty-six (18 per cent) patients had severe enough residua on leaving the service so that they had to be transferred to a Veterans Hospital for further care. These patients must be considered, at least for the present, completely incapacitated. A fair portion may be able to return to civilian life after a longer period of hospitalization. However, the great majority of this group will be invalided more or less permanently. Twelve patients were transferred to other General Hospitals for further treatment, usually some type of plastic surgery not related to their head injuries. All but one of this latter group will be able to return to civilian life on the completion of the plastic surgery. One patient was sent to a Center for treatment of the blind following a severe bilateral occipital lobe injury. It was possible to return 8 men to limited duty and 6 to full duty. This was usually at the patient's request because of his desire to remain in the service. It has been our policy to offer a discharge to a soldier with dural penetration, regardless of the degree of residua.

Two (1 per cent) patients died during their stay in this hospital. The first of these had had a brain abscess which was drained overseas. He arrived

TABLE 6

Disposition

C.D.D. or retired home	144	72.0%
C.D.D. or retired veteran	26	13.0%
Transfer to other hospitals	12	6.0%
Limited duty	8	4.0%
Full duty	6	3.0%
Mortality	2	1.0%
Convalescent center	1	0.5%
Transferred to other section or service	1	0.5%
TOTAL	200	100 %

at this hospital with a persistent draining sinus. The sinus tract was explored and was found to lead into a huge cyst in the left frontal lobe. He died about 36 hours following operation, of uncontrollable hyperthemia and status epilepticus. The second fatality was that of an officer who arrived at this hospital by aeroplane. In route he had several convulsive seizures and on arrival he was comatose. Examination showed multiple draining sinuses in the region of the vertex. He died about 72 hours after arrival, and autopsy revealed a subdural collection of pus and several small brain abscesses scattered throughout the cortex and brain stem.

Any mention of treatment has purposely been omitted from this review. The treatment of these patients at this stage has been very thoroughly discussed in many excellent papers by Woodhall and Spurling[18] and others. However, it should be stressed that the surgical repair of the defect, while important, is really a minor part in the rehabilitation of these soldiers. Physiotherapy plays an important part in helping hemiplegics recover function. Speech training is vital. Organized re-education of those patients with

such defects should be carried out. The rehabilitation of patients with dural penetration and cerebral injury is a cooperative function of the neurosurgical, neurological, physiotherapeutic and occupational therapy departments.

SUMMARY AND CONCLUSIONS

1. The late results in a series of 200 severe craniocerebral war wounds have been evaluated at the time the patients were discharged from an Army General Hospital in the zone of the interior. All of these men had sustained injury severe enough to have dural penetration.

2. It is recognized that this is not a report of end results but rather an intermediate evaluation.

3. The great majority of injuries were caused by artillery shell fragments. Over $\frac{1}{3}$ of the injuries were in the parietal regions.

4. There was evidence of infection in 47 or 23 per cent of the cases. In 15 of these there was only superficial wound infection, while in 32 there were deep or major infections, including 17 brain abscesses. There were 6 cases of extensive cerebral fungi.

The average time of debridement following injury for the whole series was 33 hours, with extremes of 2 and 504 hours. The average time of debridement in the group with infections was 25.3 hours or less than that for the series as a whole.

All patients with infection had essentially the same type of drug therapy as those that remained uninfected.

In this series foreign bodies retained in the brain seemed to have little influence on the incidence of infection.

It is felt that these statistics confirm the opinion that the most important single factor in treating penetrating craniocerebral injuries is careful and complete debridement.

5. Some type of convulsive disorder following their injury occurred in 34 or 17 per cent of the patients. Of these, $\frac{1}{3}$ had a history of convulsive seizures during the acute stage of their injury, and only 3 continued to have epilepsy at the time of disposition from the hospital. The remaining $\frac{2}{3}$ developed their first seizure from 2 weeks to 8 months after injury, and the majority of these continued to have seizures until seen at this hospital and controlled medically.

One-half (56 per cent) of the patients with convulsions had no evidence or history of infection. However, all but 3 of the remaining 44 per cent had major or deep infection. Time of debridement, unconsciousness, and retained foreign bodies seem to have little to do with the development of convulsions. This stresses the feeling that it is the cerebrodural scar and not the foreign body that is epileptogenic.

6. Twenty per cent of the patients with dural penetration had no neurological residua, and another 15 per cent had only convulsive seizures without localized residua. Motor, speech, and visual defects were the most

common. Headaches and dizziness were conspicuous because of their infrequency.

It was of interest that in 34.7 per cent of this group of severe injuries, there was no loss of consciousness.

7. There were 174 (85 per cent) patients separated from the military service, a figure which is influenced by military necessity and medical department policies, as much as by the patients' physical condition.

There were 159 (79.5 per cent) soldiers returned either to their homes or to some type of duty. These men must be considered capable of being completely or partially self-supporting. Twenty-six (18 per cent) patients had severe enough residua to necessitate further hospital care in the Veterans Hospitals. These men for the present must be considered completely incapacitated.

There were 2 (1 per cent) fatalities in this group, both occurring in patients with deep cerebral infection.

Stress has been laid on the importance of organized rehabilitation of these patients.

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COARCTATION OF THE WALLS OF THE LATERAL ANGLES OF THE LATERAL CEREBRAL VENTRICLES

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FOR MANY years my late colleague, Cornelius Dyke, and I have been intrigued by the asymmetrical appearance of the lateral ventricles of the brain in the anteroposterior encephalograms in a considerable number of cases (Fig. 1). The ventricles in these instances were usually normal in size and position, and no explanation in the clinical picture existed to account for the asymmetry. We decided, therefore, to watch the appearance of the corresponding portions of the ventricles in postmortem specimens, through the cooperation of Dr. Abner Wolf. Before anything could be done, Dr. Dyke's untimely death occurred, and the present writer undertook to make the investigation at the Jewish Hospital of Brooklyn. With the cooperation of Dr. Bernard Epstein of the Radiographic Department and Drs. Max Lederer and David Grayzel of the Pathological Department of that institution, about 64 brains, taken routinely from cases without brain tumor, and usually without any other known neurological disease, were studied.

MATERIAL AND METHOD

The brains were removed fresh and suspended in a 4 per cent formaldehyde solution in ample crockery jars, by passing a string through the basilar artery and tying the string to the lid, in order to avoid flattening or other distortion of the specimens. After a week of suspension, the specimens were removed and the fluid was allowed to drain off, after which anteroposterior roentgenograms were taken of the specimens. In many instances reasonably good pneumoventriculograms were obtained in this way. The brains were then sectioned coronally and the ventricles examined. Where asymmetry was found, sections were made for histological examination from the site where any abnormality was seen.

For the sake of objectivity, the roentgenographic and pathological studies were conducted quite independently. The result has been, however, that some of the specimens with positive roentgen evidence, if the brains were sectioned in the absence of the author, were not carefully observed, and contrariwise, some of the specimens that showed asymmetry of the ventricles on section had not had preliminary roentgen studies. Moreover, a number of the roentgen examinations were unsatisfactory in that the ventricles were inadequately visualized.

We thus ended up by having 6 cases with asymmetry of the ventricles as seen on the postmortem pneumoventriculograms, 10 cases in which devia-

tions of a similar nature were seen in the brain specimens on coronal sections; and 4 cases in which both positive roentgenographic changes and demonstrable, corresponding pathological changes were evident.

RESULTS

The Roentgenograms. The reason leading to this study was the observation that in certain clinical cases in which pneumoencephalography is done for various reasons, such as epilepsy or post-traumatic headache, the roentgenograms reveal an asymmetry of the two lateral ventricles. This consists of a

FIG. 1. Anteroposterior encephalogram showing truncated lateral angle of right lateral ventricle in an otherwise normal ventricular system.

normal looking "butterfly wing" pattern of the ventricle on one side, and a truncated one on the other side (Fig. 1). Since, usually, the "normal" side is normal also in size, the asymmetry obviously could not be explained on the basis of unilateral dilatation of this side, and the obvious conclusion had to be reached that the smaller ventricle was the anomalous one. The possible causes for this appearance we believed could be: (1) inadequate filling of the ventricle with gas; (2) a filling defect produced by a tumor mass; (3) adhesions resulting from a healed inflammatory process; or (4) a congenital anomaly.

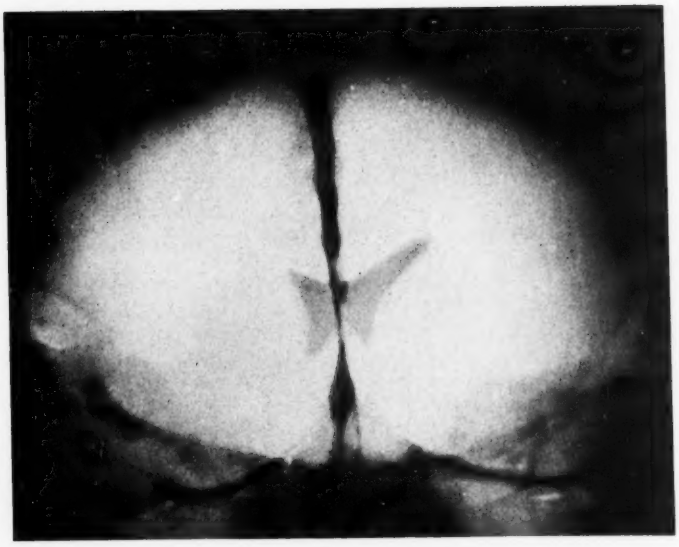

FIG. 2. Anteroposterior pneumoventriculogram of a postmortem brain specimen showing a truncated lateral angle of right lateral ventricle similar to Fig. 1.

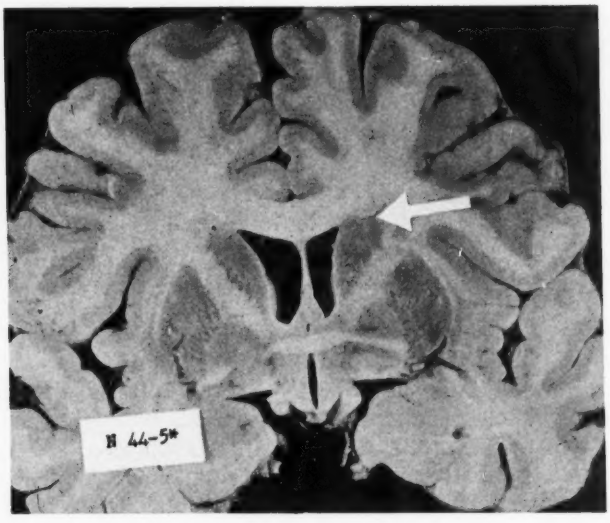

FIG. 3. Section of brain showing coarctation of lateral angle of one lateral ventricle (arrow) resulting in appearance similar to Figs. 1 and 2.

To test the first possibility, the encephalograms in which the above deviation was noted were examined carefully in lateral, anteroposterior and posteroanterior views taken with the patient both in the horizontal and vertical positions. The asymmetry was always found in all positions, thus

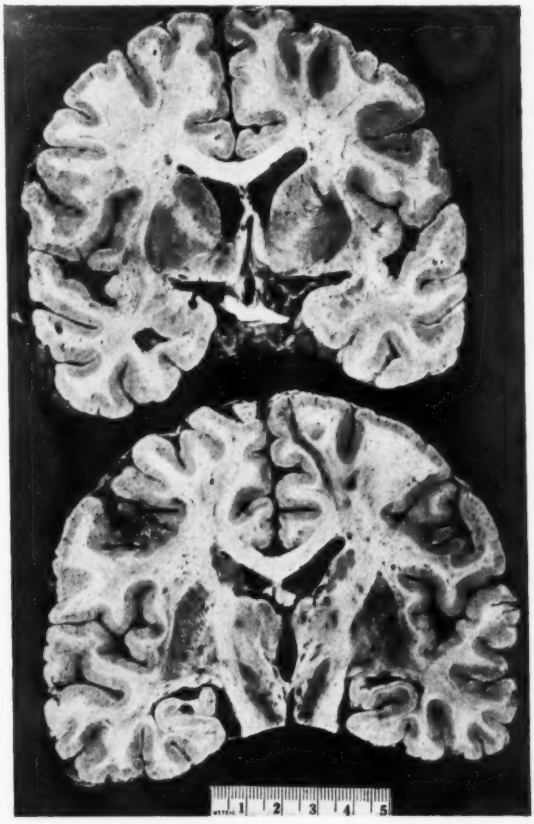

FIG. 4. Sections of brain showing coarctation of ventricular walls slightly medial to the angle resulting in small pinched-off cavity lateral to adhesions.

excluding the possibility of inadequate filling with gas as being responsible for the encephalographic appearance.

The second consideration, that a tumor may be present, was ruled out clinically by the absence of tumor symptoms and signs, and roentgenologically by the absence of displacement of the ventricular system as a whole.

The possibility that infection may have caused adhesions at these sites seemed unlikely in the absence of histories of meningitis, syphilis or diseases

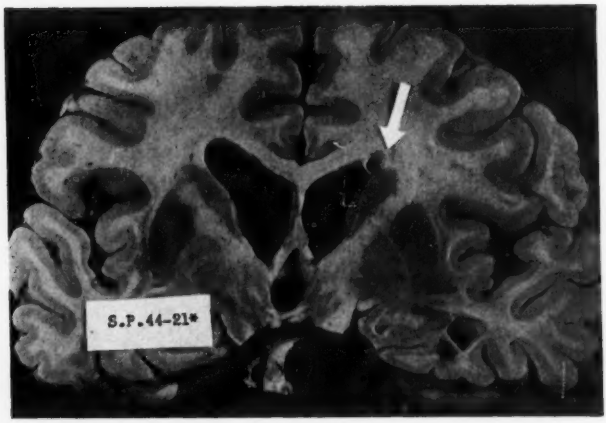

FIG. 5. Section of brain from a patient with moderate dilatation of the ventricular system showing an area of coarctation similar to that of Fig. 4 in which the adhesion was stretched out.

of the nervous system that could be interpreted as infection by viruses.

This left the fourth explanation, namely, a congenital deformity, as the most likely one, but still unconfirmed. Confirmation was forthcoming, however, in that encephaloroentgenograms from patients dying from causes other than neurological, and, if neurological, other than brain tumors, became available. These showed changes in some of the cases analogous to those seen in some of the clinical pneumoencephalograms and led to the logical conclusion that they may be due to similar causes (Fig. 2).

The Gross Anatomy. As already stated, 10 brain specimens, out of a series of 64 cases, showed asymmetrical developments of the two lateral ventricles, which on cross section presented an appearance closely resembling the encephalographic picture (Fig. 3). On close inspection there appears to be a coarctation of the adjacent walls of the lateral ventricles on the side of the smaller ventricle. In some of the specimens,

FIG. 6. Low power photomicrograph (×12) showing a lateral angle of a ventricle with coarctation similar to Figs. 1, 2 and 3. (V) Ventricle.

the adhesions were incomplete so that beyond the truncated end of the ventricle and the site of adhesion was a small cavity—evidently the most peripheral portion of lateral ventricle cut off by the adhesion from the main cavity (Fig. 4). In some cases in which there existed any degree of dilatation of the ventricle, at least on the side of the coarctation, the appearance just described changed to an elongation of the adhesion into a pillar or column within the cavity of the ventricle (Fig. 5).

Microscopic Appearance. Under the microscope, the site of coarctation is characterized by an elongation of ependymal cells between the corpus callosum and the head of the caudate nucleus (Fig. 6). These seem to show no

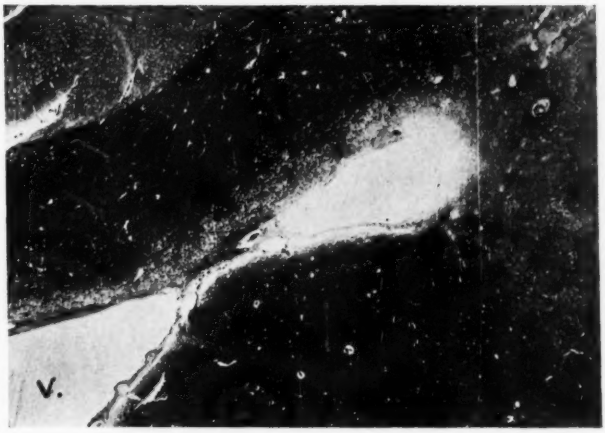

FIG. 7. Low power photomicrograph (×12) showing coarctation slightly medial to the lateral angle, resulting in a pinched-off cavity. (V) Ventricle.

degenerative changes or any tendency to cellular proliferation. In the cases in which the adhesions occur before the terminal extent of the ventricle is reached, thus leaving a portion of the ventricle cut off by the adhesion, this cut-off portion forms what looks like an ependymal cell-lined cyst, but is probably a pocket with an outlet communicating with the general ventricular cavity (Figs. 7 and 8).

No evidence of inflammatory reaction, gliosis or fibrosis was found in any of the sections examined.

DISCUSSION

In discussing this problem with Dr. Abner Wolf, who first began to watch the routine postmortem brains for these lesions, he was found to be under the impression that they occur most commonly in people who died of cardio-vascular-renal disease and are rarely seen in children. He wondered whether

cerebral edema in these cases may not cause an approximation of the ventricular walls and, in certain instances, adhesions at these sites. However, it is not seen commonly in cases of brain tumor in which the ventricle on the side of the tumor is often completely collapsed. Moreover, as we have seen in several of our specimens (Fig. 5), when dilatation of the ventricles exists the site of adhesion becomes elongated, indicating preexisting coarctation.

Finally, embryologically the lateral cerebral ventricles, which begin as rounded cavities, take on their mature shape as the basal ganglia on the floor and the fibres of the corpus callosum on the roof, develop. It is easily con-

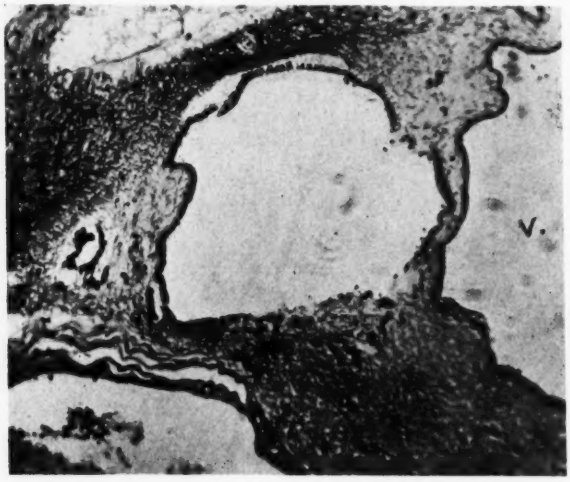

FIG. 8. Photomicrograph (×120) to show columnar appearance of the adhesion in a brain with a dilated ventricle, also to show that the pinched-off cavity is lined with ependymal epithelium. (V) Ventricle.

ceivable that the angle between these structures may form at a certain stage of development and be further sharpened as development continues to the point where occasionally coarctation of the walls takes place.

SUMMARY AND CONCLUSIONS

A certain form of asymmetry of the lateral ventricles on the pneumoencephalograms, especially in the anteroposterior views, was investigated. It was found to correspond to an area of coarctation of the ventricular walls usually at or near the extreme lateral angle of one ventricle. This was believed to take place during the developmental period of the brain.

TECHNICAL NOTES AND CASE REPORTS

BULB IRRIGATOR FOR USE IN NEUROLOGICAL SURGERY

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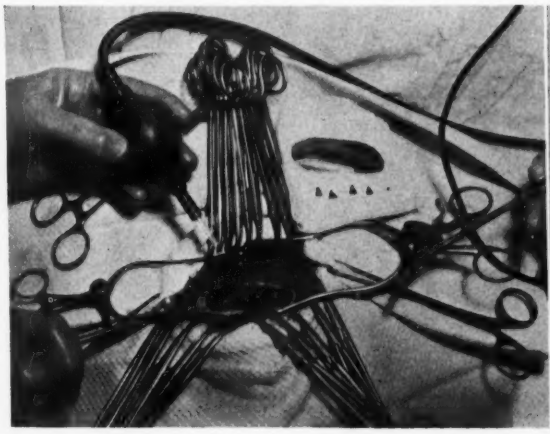
Irrigation with normal saline is extremely useful in operations involving brain, spinal cord, and peripheral nerves, in that it keeps nervous tissue moist, washes away blood clot, affords better visibility, and cleanses traumatic wounds.

Various types of irrigators have been used, such as glass syringes, glass syringes with attached rubber bulb, and rubber bulb ear syringes, the latter having the advantage of being unbreakable.

This communication describes a rubber bulb irrigator (Fig. 1) which has proved quite satisfactory in a neurosurgical center during the past year. It consists of a rubber bulb, similar to a sphygmomanometer bulb, having a simple valve in each end allowing fluid to pass in one direction only. To the outlet valve is attached a short piece of rubber tubing containing a small glass or metal tube, and to the intake valve is attached a length of rubber tubing con-



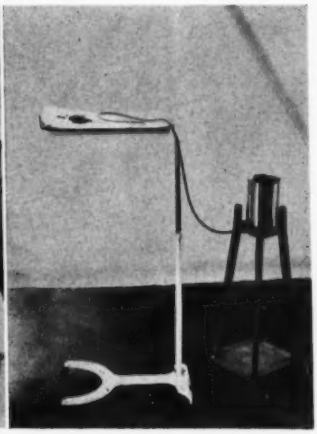


FIG. 1 (*left*). Bulb irrigator illustrating glass tip attached to the outlet valve and rubber tubing connected to the inlet valve, being used during a cranioplasty.

FIG. 2 (*right*). Bulb irrigator with rubber tubing connected to an irrigating can placed on a wooden stand at a level lower than the operative field. Mayo stand shown to demonstrate the approximate height of operative field. (Sterile covers for stand and irrigating can not illustrated.)

nected to an irrigating can filled with normal saline. Slight pressure applied to the bulb delivers an irrigating stream; and when pressure is released, the bulb automatically and rapidly refills from the irrigating can.

The entire apparatus is sterilized by boiling and is connected to the autoclaved irrigating can by the scrub nurse. The can containing the bulb with attached tubing is then placed on a draped wooden stand (Fig. 2) at a level lower than the operative field, near the operating

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table, and covered with a folded sterile towel. After the table is draped, the circulating nurse lifts the sterile towel to permit one of the operating team to remove the bulb with attached tubing and place it on the operating stand or instrument tray, the tubing being attached to the drapes in the same manner as the suction tubing. The saline is replenished by the circulating nurse whenever necessary. A sterile dairy thermometer is kept in the irrigating can in order to afford a temperature of 100° to 103°F.

Initial filling is obtained by pressure on the bulb, which when released will draw fluid up from the can, filling the bulb and displacing the contained air. The bulb being used at the present time (Fig. 1) has a volume of 50 cc.

The force of the stream is regulated by the amount of pressure on the bulb. If constant irrigation is desired, raising the irrigating can higher than the operative field will cause a constant flow of fluid from the tip of the bulb due to siphonage, the pressure being dependent upon the height of the can.

Valves of various types have been used consisting of a metal T, rubber T, metal ball, blood pressure valve, etc. However, a simple thin rubber disc, cut from an ordinary rubber glove, has proved most satisfactory. Details of the bulb irrigator, particularly the valve, are further described in the illustrations and accompanying legends (Fig. 3).

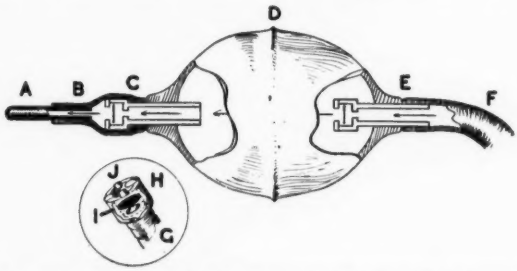

FIG. 3. Sketch of bulb irrigator. A, Hollow glass or metal tip (diameter of bore approximately 1 mm.) attached by a short piece of tubing, B, to the outlet valve, C. D, Rubber bulb having an opening at each end containing valves, C and E. E, Inlet valve connected to rubber tubing, F, which is attached to irrigating can. Inset shows oblique view of valve (C and E) which consists of a small tube, G, one end of which has a larger bore, H, containing a small, thin rubber disc, I, retained by four prongs, J, bent towards the center of the tube.

Since the bulb is usually placed at a higher level than the irrigating can, preventing a constant forward flow due to siphonage, there is a reversed siphon effect which attempts to draw fluid from the bulb back to the irrigating can. In a few of the valves first tried this occasionally occurred, allowing air to enter the outlet valve, replacing a small amount of fluid in the bulb. This was easily corrected by inverting the bulb and, by pressure, evacuating the air as in the initial filling. However, the valve illustrated has been found to prevent this leakage. The thin rubber valves will withstand many sterilizations but will require replacement in due time.

A holder for the irrigating can can be made to attach to an intravenous stand and levels adjusted as desired. However, the writer has found the wooden stand to suffice for all operative procedures.

The advantages of this bulb irrigator are many. It is a closed system, eliminating long exposure of irrigating fluid to airborne bacteria. Space on the instrument tray is increased by avoiding an additional basin for saline. Time is saved by the rapid filling of the bulb as contrasted to the slow process of passing and filling an ear or glass syringe. The deep irrigating can facilitates maintenance of correct temperature. The scrub nurse is relieved of the before-mentioned details, allowing more time for other duties. The outer surface of the bulb is usually dry which helps prevent accidental dropping. Convenience of the bulb has been a very important factor to the operating team.

A rubber catheter can be attached to the tip of the bulb irrigator and deep tracts in lacerated brain wounds can be well irrigated from the depth of the wound. In neurorrhaphies constant drops of saline can be directed on the nerve ends to be sutured, washing away blood, increasing visibility, and with only a minimum of effort on the part of the assistant (Fig. 4). Photography in the operating room has been improved by irrigating the operative field with the bulb while the photograph is being taken, washing away blood from oozing points.

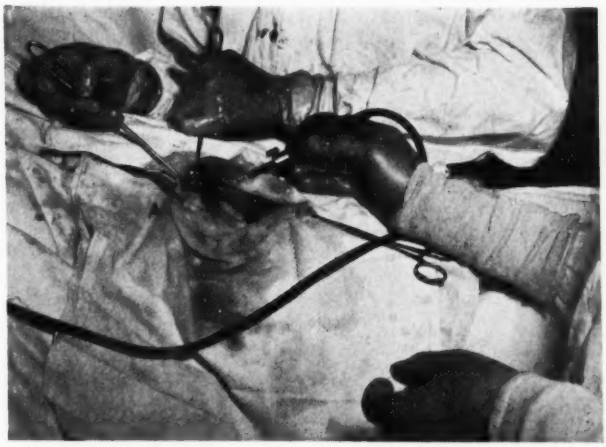

FIG. 4. Bulb irrigator (earlier type using B.P. valve at outlet end) being used in peripheral nerve surgery. Note catheter, A, in operative field which is connected to suction tip affording constant removal of excess irrigating fluid.

To avoid excessive wetting of drapes, a catheter connected to the suction tip and placed in a dependent portion of the operative field has been found useful, in that the fluid is removed automatically. Various metal tips with multiple openings, such as a duodenal tube bulb, have been used and found more satisfactory than a catheter.

SUMMARY

1. A bulb irrigator with rubber tubing connected to an irrigating can is described for use in neurosurgical operations.
2. Slight pressure applied to the bulb delivers an irrigating stream; and, when pressure is released, the bulb automatically refills through a tube connected to the irrigating can.
3. The advantages of the described bulb irrigator are discussed.

ARTERIAL ANGIOMA

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(Received for publication November 15, 1945)

In a recent publication[1] we made an attempt to classify the surgical vascular diseases of the brain. We referred to the congenital vascular diseases as angiosis, with the following

sub-classifications: (a) Cavernoma; (b) Telangiectasis; (c) Sturge-Weber-Dimitri disease; (d) Angioma—angioma arteriale—angioma venosum; (e) Arteriovenous aneurysm.

Our study was based on personal experience with the above-mentioned diseases, with the exception of arterial angioma. As is well known, from the time of Virchow[7] to the present there has always been discussion as to whether or not there is a true or pure form of arterial angioma. Some authors have used the term arterial angioma to describe those lesions having a greater number of arterial vessels in relation to the venous vessels (veins). Others, such as Harvey Cushing,[4] gave this name to arteriovenous aneurysm. According to our definition, arterial angioma must consist entirely of arterial vessels and, at times, of undifferentiated vessels. Furthermore, it must be localized in some part of an artery. The arteries may be afferent or efferent. Bergstrand[2] agrees with this last statement.

The symptoms of patients suffering with this illness are undulant. Every time there is a rupture of the lesion a hemorrhage occurs, accompanied by neurological symptoms depending on the location of the aneurysm. An examination will reveal a systolic bruit and roar. As we are dealing with malformation in cases of this type, other vascular abnormalities will in all probability also be present. In our case the patient had an arterial angioma on his left cheek and conjunctiva. These signs enabled us to make a tentative diagnosis before postmortem verification.

We were able to find only three verified cases in the literature: One was reported by Casper,[3] one by de Martel, François and Guillaume,[6] and one by Leeser.[5]

In the following typical case of arterial angioma, an intracerebral vascular malformation was also present, the patient having a double basilar artery.

CASE REPORT

Chart No. 2193. M.S.M., a married man, aged 63, was admitted to the Neurological Institute on Jan. 31, 1945. His family and personal histories were not unusual. In 1923 there was sudden onset of intense headache accompanied by stiffness of the neck, nausea and vomiting, pain over the region of the heart, and dizziness. He was hospitalized 10 days. In 1924 and thereafter every 2 or 3 years until 1941 similar attacks occurred, obliging the patient to be hospitalized for short periods. In 1941 he had an attack, but this time he lost consciousness for 6 hours, and he remained in the hospital for 18 days. The last attack occurred in January 1945, and was accompanied by epistaxis.

FIG. 1. Angioma of the face.

On admission the symptoms were suggestive of meningitis. A lumbar puncture revealed a hemorrhagic fluid. There was stiffness of the neck. Kernig and Brudzinski signs were positive; radial and cubital reflexes were abolished; there were slight abdominal and cremaster reflexes; reflexes of patella and Achilles tendon were absent. The patient was semiconscious.

On Feb. 13, 1945, he was transferred to the neurosurgical service, as it was suspected that he had a vascular lesion and arteriography might be advisable. On examining the face of the patient slightly raised areas of the skin, 1.5 cm. in diameter, were noted in the left frontal region, left cheek and half of the upper left lip. These areas were palpable and soft to touch. There was also a quick pulsation on the left cheek, which when palpated could be detected as a definite thrill pulsation. The skin appeared slightly thickened, the pores pronounced or enlarged, and wine-colored in comparison with the rest of the face (Fig. 1). In the

conjunctiva of the left eye could be seen enlarged blood vessels, reddish-wine in color, which gave the impression of a tumor or angioma of the eye. No bruit of the eyeball could be heard nor was there pulsating exophthalmos. Severe psychic disturbances with stupor were present. Neurological examination of the cranial nerves was negative, although weakness was present on both sides. The tests of skin sensitivity and pain were difficult due to the patient's weakened condition and mental state. The reflexes of the superior extremities were symmetrically diminished. Hoffmann's test was negative on both sides. Examination of thorax, abdomen and extremities revealed no alteration of importance. Blood pressure, right arm, 100. Cardiovascular system normal. Lumbar puncture gave a xanthochromic fluid with hemorrhagic sediment: 0.45 gr. of protein 0/00; Nonne and Pandy tests ++; 0.12 cells per cc. Uremia 0.70. Kahn, negative.

The patient continued to grow worse. During the evolution of the attack he suffered intense pain in the right side of his face. On March 4 he went into coma, and died on March 5.

FIG. 2. Abnormal and normal carotid arteries.

Autopsy. The left common carotid, and internal and external carotid arteries were all larger than the right (Fig. 2). This enlargement was more pronounced in the facial and internal maxillary arteries. There was a soft tumorous growth formed by a number of tortuous dilated arterial vessels. The left internal carotid opening at the base of the skull was larger than that on the right. The dura was normal and arachnoid thick. The surface of the brain was yellowish, with scattered brown pigmentation, especially in the frontal, temporal and occipital poles and at the base in the interpeduncular spaces. The arachnoid was extremely thickened in both Sylvian fossa and basal cistern in the vicinity of the optic chiasm. The left internal carotid was twice as large as the right (Fig. 3). The anterior communicating artery was larger than the internal carotid. The Sylvian and left posterior communicating arteries were also enlarged (Fig. 4). The right posterior communicating artery was almost non-existent. The basilar artery was replaced by two parallel, mesial arteries which appeared

FIG. 3. Anterior view. (1) Left internal carotid, double in size. (2) Anterior communicating artery, larger than internal carotid. (3) Bunch of vessels.

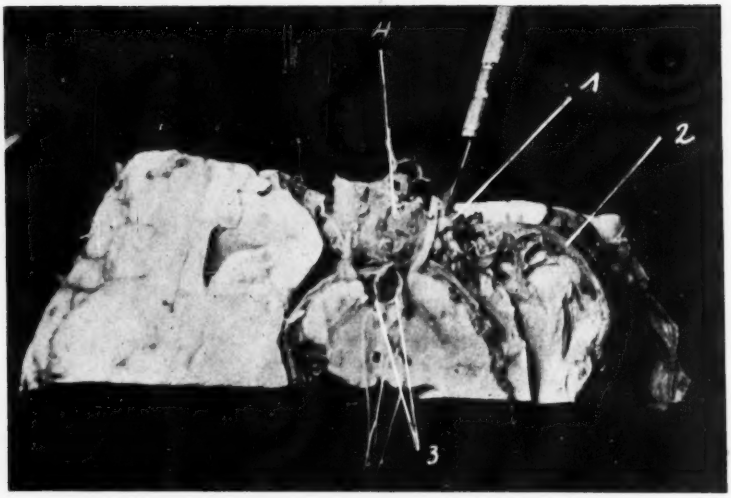

FIG. 4. Section through the brain stem. (1) Left posterior communicating artery. (2) Left Sylvian artery. (3) Two mesial arteries replacing basilar. (4) Bunch of vessels from chiasm to hippocampal fissure.

to be prolonged branches of the vertebral arteries. The inferior aspects of the chiasm and left optic tract were replaced by a bunch of tortuous vessels of different sizes. This mass of vessels spread along the optic tract to the hippocampal fissure, and enveloped the left choroidal artery. The left superior side of the optic tract was also occupied by these abnormal vessels, which were larger than those in the interior face of the optic tract. The walls of these vessels

FIG. 5. Vessels of chiasm varying in size and diameter. (×40)

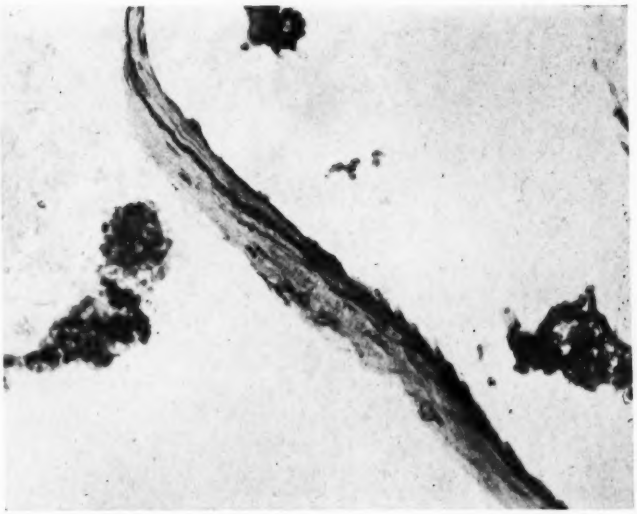

FIG. 6. Vessel with thin wall, in which elastic fibrils may be seen (arterial). (×400)

were of varying thickness and adherent to arachnoid. Brown pigmentation was observed among the adhesions. Vessels as well as arachnoid were yellowish. Where the Sylvian artery rises, there was an abnormal, extremely tortuous vessel, 5 mm. in diameter and 10 mm. in length. No veins were found along the vascular surface. This bunch of vessels started in the

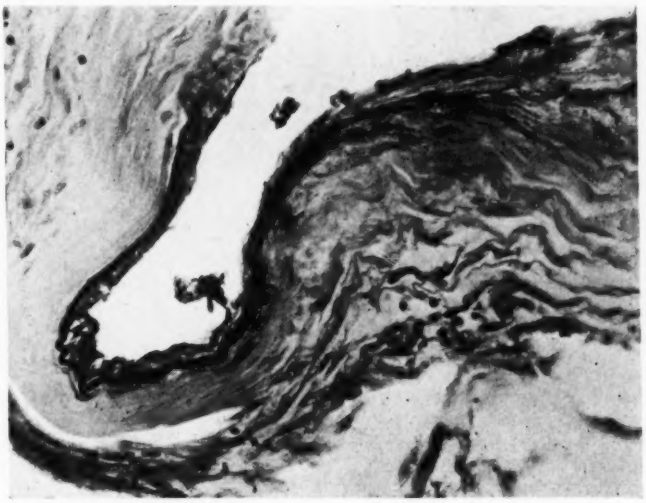

FIG. 7. Median-caliber arterial vessel. Elastic layer is easily identified; at upper right this layer splits into fibrils. (×400)

left internal carotid, occupied the whole chiasmatic and interpeduncular region and was continued by the posterior vessels of the circle of Willis. The left ventricular system was more enlarged than the right, and the ventricular walls were slightly brownish. Cross sections of the brain revealed no injuries or oedema.

Microscopic Examination: Dr. Julio Espinosa. The specimen was a cross section of the angioma and a small portion of the optic chiasm. Some of the vessels varied in size from 0.10 to 1 mm. in diameter and invaded the chiasm (Fig. 5). Most of these vessels had a wide lumen and thin walls, in which connective and elastic tissues could be seen. Elastic tissue was present in every vessel, even in those with thinner walls (Fig. 6). Generally no definite layers formed by elastic fibrils were observed. Many of them appeared to be split (Fig. 7) while others tended to form bundles. Elastica interna in wave shape is rarely seen in median-caliber arteries. The interstitial tissue was composed of chiasmal nerve tissue, which in places was degenerated and somewhat oedematous. The pia was oedematous, and hemorrhagic foci with brown pigment were found. There were also inflammatory islands with lymphocytic perivascular infiltration.

SUMMARY

This appears to be a typical case of arterial angioma of the brain in an individual who presented malformation of the blood vessels of the face and *Willis poligono*. The macroscopic and microscopic findings confirm the diagnosis, since the majority of the blood vessels were revealed to be arteries or undifferentiated vessels. Remains of old hemorrhage as well as of chronic arachnoiditis were evident. This report proves the existence of true arterial angioma.

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NERVE STIMULATION IN PARAPLEGIC PATIENTS BY MEANS OF BURIED INDUCTION COIL

PRELIMINARY REPORT

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With a view to initiating useful muscle activity in paralyzed patients unable to walk without artificial aid, an electrical device has been worked out to initiate automatic step-page. While this principle of stimulation is not new, its present application is.

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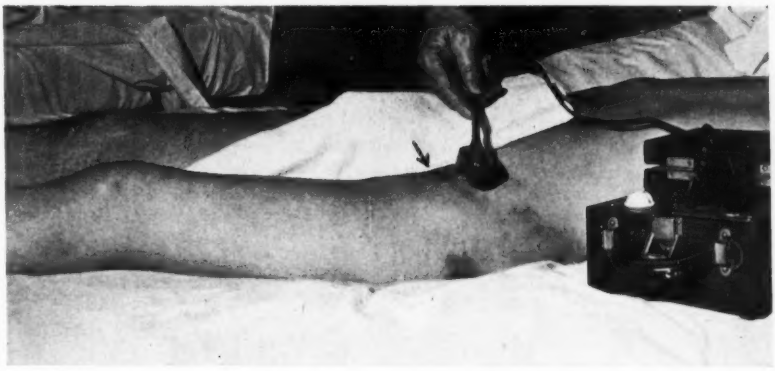

FIG. 1. Left leg relaxed on bed. The primary inductance coil is being held by the examiner about 1 inch above the skin. To the right is the box containing buzzer, flashlight batteries, and push-button. Projected refinements will permit the use of a far more compact battery box and a smaller primary coil. The arrow indicates the healed incision over the femoral nerve. Beneath the small protuberance of skin lateral to the incision lies the buried secondary coil, approximately $\frac{1}{3}$ the size of the primary.

This device consists of a small inductance coil, $\frac{3}{4}$ inch in diameter and $\frac{1}{4}$ inch thick, buried beneath the skin. Two insulated wires lead to electrodes which are attached to the surface of the desired nerve.* A primary inductance coil outside the skin induces a current in the buried secondary coil.

The secondary coil is wound with 2400 turns of No. 40 enamel insulated copper wire, in the center of which is a core of soft iron to improve its efficiency. The primary coil is wound with fewer turns and heavier wire, and is also provided with an iron core. Both coils can be made still smaller and remain effective, since the present preliminary tests indicate that a

FIG. 2. Beginning contraction of quadriceps muscles (arrow) as button is pushed; 4.0 volts supplied to primary coil. This initial extension of the leg was followed by flexion of the thigh on the pelvis.

* Special thanks are due Chief George J. Barrett and Radioman First Class J. Lifford, U. S. Coast Guard Training Station, Atlantic City, New Jersey, for their cooperation in constructing, testing and charting the wave forms of the apparatus.

large margin of reserve exists; that is, the primary coil is effective at a distance of approximately one inch from the surface of the skin, with a current of less than half of what it can carry. It might be added that the buzzer used to convert direct into alternating current can easily be replaced by a much smaller device, such as that used in radio sets. Thus the whole apparatus could be made so compact as to be worn on the patient's belt.

The buried coil and wires are completely insulated by a coating of plexiglass. (Similar plastics have been used and implanted permanently in the human body, notably acrylic plates in skull defects.) The electrodes themselves consist of nichrome steel wire.

Should this "closed" method of nerve stimulation continue to show promise, it might be applied not only to peripheral nerves but to selected nerve roots of the cauda equina, as first applied by the author (prior to the past war) in laboratory tests. A special leg brace has been designed for use with this method of nerve stimulation. The brace is hinged at the knee

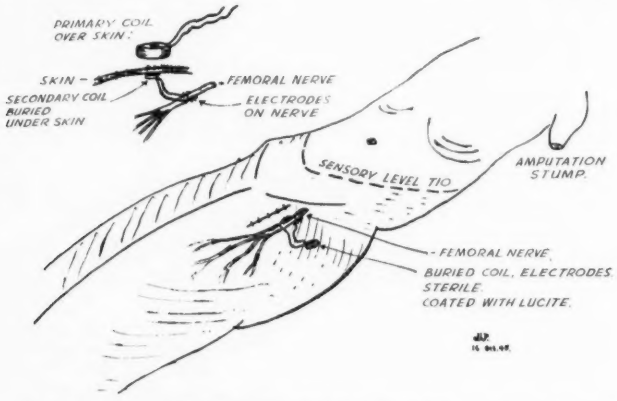

FIG. 3. Diagram showing arrangement of primary and secondary coils and attachment of electrodes to femoral nerve.

joint and provided with a simple automatic lock to prevent buckling or excess flexion of the leg.

In the present instance, a patient was selected with a paraplegic level at T-10. His left arm was amputated near the shoulder, so that he could not use a left crutch. Walking in braces was also rendered impossible because of extensor spasms in both legs, especially the left. With help in flexion of the left thigh, however, it was felt he might be able to walk. Hence the buried inductance coil was attached to the left femoral nerve and the coil itself implanted at operation beneath the adjacent skin. The implanted coil and its wire leads were sterilized in Bard-Parker solution for 17 hours, 70 per cent alcohol for 4 hours, and were then immersed in sterile saline solution for 1 hour.

Direct contractions of the left quadriceps and indirect contraction of the left iliopsoas muscles can be induced at will by proper placement of the external primary inductance coil, or by regulation of the current flowing through it. When the patient is supported in a standing position, satisfactory flexion of the leg and thigh can be electrically initiated to a 45-degree angle from the straight vertical position of the leg. There has been no appreciable change in these induced electrical effects during the 4 months the coil has been implanted.

Preliminary tests also demonstrate an effect of reflex spread of induced stimulation, resulting in dorsiflexion of the left toes and foot, which should benefit an automatic gait. A secondary finding was the demonstration of "Sherrington phenomena," such as ipsilateral facilitation and crossed extensor thrust in the opposite leg, which may also prove beneficial in automatic walking.

While it is too early to appraise the usefulness of this device, it is presented to stimulate thought along physiological lines for the rehabilitation of these tragic cases. If the method proves successful, selective alternating stimulation of various nerves may well prove of benefit to paraplegic patients, once their spastic contractions have been toned down.

SUMMARY

A method is presented for "closed" electrical stimulation of a peripheral nerve in a paraplegic patient by means of a small buried inductance coil, controlled by the patient. This method is designed to facilitate walking, when aided by a special brace.

PARAVERTEBRAL INJECTION OF PROCAINE FOR PAIN PRODUCED BY AORTIC ANEURYSM

CASE REPORT

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(Received for publication January 9, 1946)

Although paravertebral injection of procaine and/or alcohol has become a recognized procedure for the relief of pain produced by aneurysms of the thoracic portion of the aorta, the reason for this relief, as well as the actual mechanism by which the pain is produced, is the subject of controversy. In previous cases reported by White,[4] unilateral pain produced by aortic aneurysms was relieved by paravertebral injection of the corresponding side, and the relief was attributed to interruption of the afferent fibers from the damaged portion of the aorta. In the case to be presented here, the pain was substernal and not lateralized, yet prolonged relief was afforded, first by paravertebral injection of procaine on the left side and subsequently again by a similar injection on the right side. The case is reported for its implications concerning the mechanisms involved both in the production of the pain and in the relief of the pain by a temporary paralysis of the regional sympathetics.

CASE REPORT

1st Lt. Liu Ku Ming, Chinese Army in India, 25 years of age, first became ill in January 1944 while on active duty with his unit in Burma. He noted gradual onset of substernal pain, hoarseness, persistent cough, dyspnoea on exertion, and occasional hemoptysis. In May he was admitted to a hospital in the forward area and one month later was transferred to a base hospital. The pertinent laboratory findings here were a positive blood Wassermann and X-rays of the chest which showed a marked enlargement of the heart and an aneurysm of the arch of the aorta. During his 6-month stay in this hospital the persistent substernal pain necessitated 1 to 5 daily hypodermic injections of morphine (usually gr. 1/6), except for an occasional good day. During August and September cardiac decompensation developed and then disappeared on supportive therapy. In October he was started on an anti-syphilitic regime of potassium iodide and bismuth, without producing any change in his chest pain.

On Oct. 6, 1944 he was transferred to our hospital with the same principal complaint of vague, gnawing substernal pain which varied in intensity, being relatively slight at times and then again quite severe. There were marked hoarseness of the voice and bilateral absence of the pupillary reaction to light. Examination of the cardiovascular system showed a definite tracheal tug, a harsh systolic murmur over the entire precordium, left axilla, left interscapu-

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lar area and right subclavicular region, and a softer diastolic murmur over the 2nd right interspace at the sternal border. The blood pressure was 130/40 in each arm. There was a characteristic Corrigan pulse but no exaggeration of the capillary pulse was visible. The spleen was moderately enlarged; the liver was at the costal margin and was not tender. There were a few rales over the base of the left lung. Fluoroscopy and X-rays of the chest revealed a markedly dilated heart with an aneurysm of the arch of the aorta which displaced the lower segment of the trachea to the right. There was evidence of a small amount of fluid at the base of the left lung and slight erosion of the anterior surfaces of the bodies of the 5th and 6th thoracic vertebrae. There was no percussion tenderness over the sternum or spine.

During the first week under our observation an effort was made to stop the administration of morphine, and, at the expense of some discomfort to the patient, it was reduced to 1 or 2 doses daily. He remained in bed most of the time and took little part in the ward activities. The administration of potassium iodide and bismuth was continued. On Dec. 14, 1944 a paravertebral injection was done according to the technique of White and Smithwick,[5] and 15 cc. of 1 per cent procaine were injected along the left side of the body of the 1st thoracic vertebra. A second needle was then inserted on the left down to the 2nd thoracic vertebra, but freely flowing, non-pulsating arterial blood appeared, so the needle was withdrawn. A left-sided Horner's syndrome and a sympathetic paralysis of the left arm appeared within a few minutes, and there was no untoward reaction to the procedure. The substernal pain was completely relieved for 2 hours and then returned with considerably reduced intensity. During the next week there was a striking increase in his activity about the ward and he complained of only mild and intermittent pain. He received morphine twice during this first week, each time at night, and 4 times during the next 2 weeks. He then remained free from pain, and after several weeks asked to be returned to light duty. While this was being arranged he developed a severe upper respiratory infection on February 5, which lasted for 2 weeks. Following this the substernal pain returned as before. On Mar. 7, 1945 the paravertebral injection was repeated, this time, however, on the right side; 15 cc. of 1 per cent procaine were injected alongside the 1st thoracic vertebra and 15 cc. alongside the 2nd thoracic vertebra. A satisfactory right Horner's syndrome and sympathetic paralysis of the right arm was obtained. There was again immediate relief of pain and the patient was discharged to limited duty one week later, still free from pain.

On June 5, 1945, 6 months after the left-sided injection and 3 months after the right-sided injection, he returned for reexamination. He had remained free of pain and able to carry out his assigned duties with his unit. There was no significant change in his general physical examination or in the examination of his cardiovascular system. X-rays of the chest showed disappearance of the density at the base of the left lung, a slight decrease in the size of the heart shadow, but no change in the aneurysmal shadow.

COMMENT

The interesting features of this case are, first, the relief of midline pain by paralysis of the fibers of the upper thoracic portion of the sympathetic nervous system of the left side and subsequently of the right side, and secondly, the prolonged relief which followed the temporary paralysis produced by the injection of procaine.

It seems fairly definite, from the deep-seated and midline location of the pain and the lack of local tenderness over the sternum and spine, that the painful impulses in this case were arising in the aorta rather than as a result of pressure of the aneurysm upon the thoracic cage or intercostal nerves. Since visceral afferent fibers from the arch of the aorta are believed to reach the spinal cord via both the right and left sympathetic chains, the simple fact of blockage of the afferent impulses of one side does not adequately explain the relief of pain. Injection of each side, singly, resulted in relief of pain; thus it is reasonable to assume that nerve impulses concerned with the production of the pain were traversing both sympathetic chains. Yet interrupting the impulses of one side was sufficient to stop the pain.

The persistence of relief from the pain long after the procaine had been absorbed also de-

mands a more elaborate explanation than the simple blocking of afferent impulses. Such a response to procaine has been noted frequently in the literature for a variety of painful conditions and was present in two of the three cases of aortic aneurysm reported by White.[4] White's Case 1 experienced relief of pain for 36 hours after the preliminary paravertebral injection of procaine, and Case 2 was free from pain, except for the 5th and 6th weeks, for 2½ months after the injection of procaine. In causalgia a similar relief of pain frequently follows paravertebral injection of procaine, and the question may be raised as to whether the fundamental mechanism actually producing the pain may not be essentially the same.

In a recent discussion of the causalgic states, Livingston[2] has suggested that the irritative impulses originating in the injured structure may under certain conditions produce a pathologic pattern of excitation in the gray matter of the corresponding segments of the spinal cord, which tends to become self-sustaining and, by stimulation of the adjacent lateral and anterior motor horns, to produce peripheral changes, which in turn become the source for additional pathologic sensory impulses and thus further contribute to the central dysfunction. The presence of hyperesthesia associated with the pain in some cases of aortic aneurysm may be evidence of some such 'vicious circle.' Livingston further postulates that the cure of causalgia and related states by interruption of the sympathetic nervous system, excision of damaged segments of arteries or local peripheral injection of procaine may be due to the breaking of the vicious circle as a result of the temporary cessation of the abnormal afferent impulses.

If this theoretical concept be applied to this case, an explanation is available for the relief of the midline pain by a unilateral injection and for the prolonged relief produced by the procaine. Interpreted in this light, the temporary reduction of the visceral afferent impulses from the aorta as a result of the unilateral paravertebral injection was sufficient to break the vicious circle and thus give relief from the pain.

This case also gives clinical corroboration of the bilateral innervation of the arch of the aorta and of the pathway of the visceral afferent fibers to the spinal cord via the upper thoracic segments of the sympathetic chains.

Recent usage of slowly absorbed anesthetic agents for paravertebral injection (Lee, Macht, and Pierpont[1] and Rasmussen and Alessi[3]) suggests the possibility of increasing the effectiveness of these injections in relieving selected painful conditions without the addition of the destructive action of alcohol.

While in our patient the 6-month history of morphine administration might be considered a complicating factor in evaluating the results, the contrast in the ease with which the drug was stopped after the first injection and the difficulty when it was attempted before, the lack of withdrawal symptoms, and the fact that the patient's behaviour about the ward corroborated his statements as to the presence or absence of the pain, seemed sufficient verification of the genuineness of the relief from pain.

SUMMARY

A patient with an aneurysm of the arch of the aorta producing midline substernal pain is presented. Paravertebral injection of procaine on the left side (1st thoracic vertebra) resulted in relief of pain for 3 months. Upon return of the pain a similar injection of procaine on the right side (1st and 2nd thoracic vertebrae) likewise produced prolonged relief of pain. The interpretation of these results is discussed in the light of recent theories of the mechanism involved in the production of the pain of causalgic and related states.

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A TECHNIQUE OF NERVE SUTURE*

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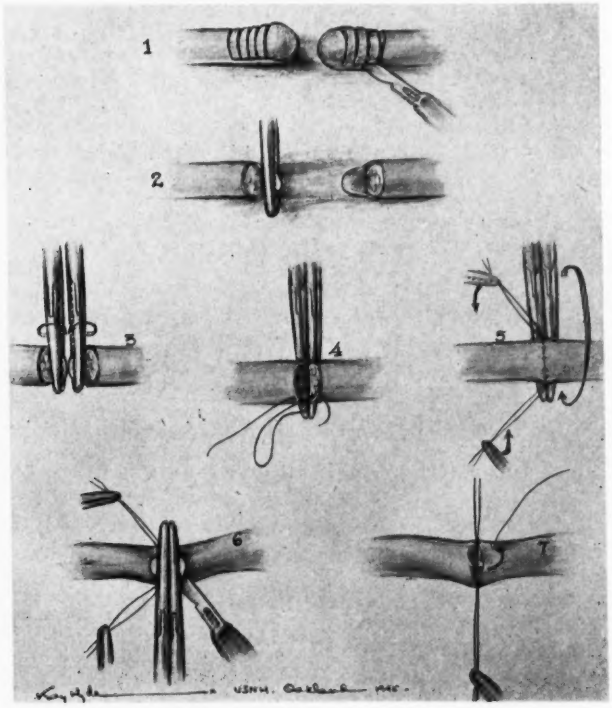
From a technical standpoint two essential factors in the optimal suture of a divided peripheral nerve are stabilization and lack of tension of the nerve ends during the suture procedure. We have devised a method of suture that fulfills these requirements. The method is illustrated by the accompanying drawings.

The neuromas are cut back serially using a straight surfaced blade until satisfactory bundle patterns are obtained, leaving a small lip of sheath extending distally from the end surface as shown in Fig. 1. In cutting back the proximal and distal nerve ends the bundle pat-



* The opinions expressed in this article are the private views of the authors and are not to be construed as official or reflecting the views of the Navy Department.

terns can be compared and the holding tabs of nerve sheath placed so that corresponding end bundles will be brought into opposition during suture.

Small matched mosquito clamps are then placed on the tabs exactly adjacent to the end surface of the cut nerve (Fig. 2). The clamps are applied with handles away from the operator to be held by the first assistant. The two clamps are then rotated horizontally 180° (Fig. 3), bringing the end surfaces of the nerve into accurate approximation (Fig. 4). The assistant holds both clamps in one hand while suture is carried out. Nearly two-thirds of the surface of the nerve can be sutured from this position. The most medial and most lateral sutures are left long for use in rotating the nerve to suture the posterior surface (Fig. 5). The medial long end is passed beneath the nerve and the two clamps are rotated as one toward the the surgeon (Fig. 5), presenting the posterior surface of the anastomosis (Fig. 6). The small sheath tabs are then removed by cutting directly along the holding edge of the clamp. With the two long-ended sutures stabilizing the nerve (Fig. 7), the remaining posterior surface is sutured. The two long suture ends are cut and the nerve is allowed to rotate back into normal position.

With this method the nerve ends are stabilized in accurate approximation without tension during the entire suture procedure. The surgeon is not crowded either by his assistant or by instruments interfering with his field. It is possible to place sutures more rapidly and precisely and without trauma to the nerve ends.

This technique has been employed in all end-to-end sutures done in this clinic during the past 12 months. In our experience it has enabled us to accomplish more meticulous suture in all cases, and has greatly facilitated suture in cases in which approximation would otherwise have been extremely difficult.

NOTICES

THE AMERICAN BOARD OF NEUROLOGICAL SURGERY

During the war, in order to cooperate with the Surgeons General of the Army and Navy in the classification of specialists, The American Board of Neurological Surgery permitted applicants whose training was satisfactory to take their examination before complying with the requirement of two years independent practice of neurological surgery. In no instance, however, was the Board's certificate issued until all requirements had been fulfilled. At its last meeting in Nashville, Tennessee on April 7, 1946, the Board voted to return to its prewar practice and in the future no candidate will be examined until he has complied with all requirements of the Board both as to training and practice. However, the Board invites prospective candidates to submit their qualifications to the Board when their period of training is completed and before they have begun their period of practice.

SECOND SOUTH AMERICAN CONGRESS OF NEUROSURGERY

The second South American Congress on Neurosurgery will be held in Santiago, Chile, in April 1947.

The following subjects have been assigned to the various neurosurgical groups in Argentina, Brazil, Chile, Peru and Uruguay:

1. Hydrocephalus: (a) Classification, Peru; (b) Pathology, Argentina; (c) Clinical aspects, Brazil; (d) Treatment, Argentina.

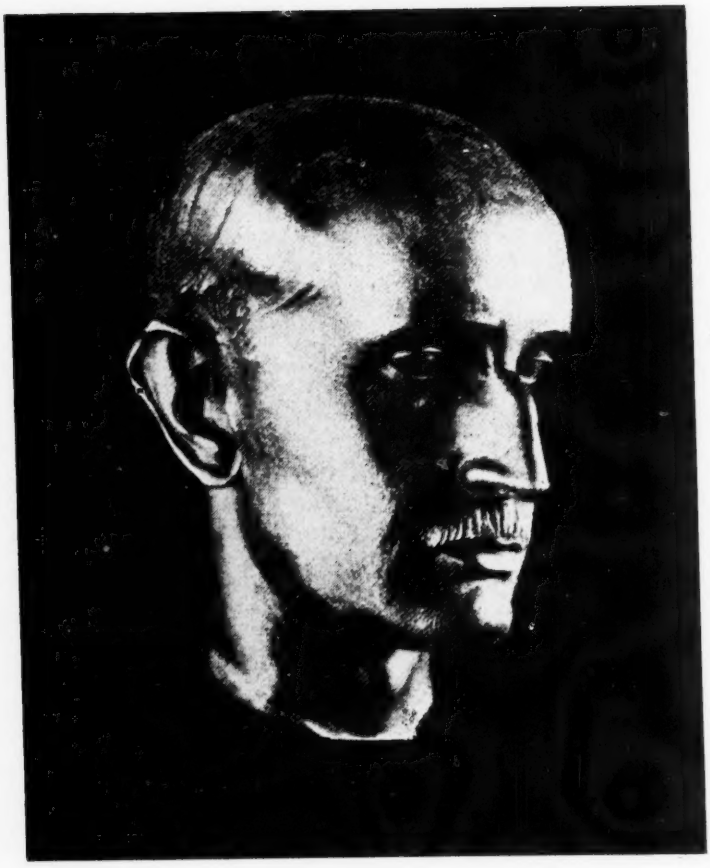
2. Histology of Brain Tumors in Relation to Clinical Symptoms: (a) Pathology and clinical course, Argentina; (b) Pathology and surgical treatment, Argentina; (c) Pathology and radiotherapy, Uruguay.

3. Brain Abscess: (a) Pathology, Uruguay; (b) Clinical aspects of cerebral abscess, Brazil; (c) Clinical aspects of cerebellar abscess, Argentina; (d) Treatment, Chile.

The program will also provide time for open discussions, practical demonstrations and moving picture projections.

Inquiries may be sent to: Dr. Alfonso Asenjo, Director, Instituto Central de Neurocirugía y Neuropatología, Pabellón Cushing, Santiago, Chile.

JOURNAL OF NEUROSURGERY



PÍO DEL RÍO-HORTEGA, BY HIS CLOSE FRIEND THE SPANISH SCULPTOR JUAN CRISTÓBAL.
(From *Residencia*[1])

PÍO DEL RÍO-HORTEGA
1882–1945*

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Pío del Río-Hortega was born on May 5th, 1882, in the town of Portillo near the University city of Valladolid,‡ once the capital of Castile. His mother died when he was five years old, leaving a family of eight children. Very little is known of the childhood and early youth of del Río except that he was brought up by his three older sisters in the family's castle at Portillo. His father was a respected Castilian *hidalgo* who eventually moved to Valladolid in order to take his family to the schools and to the ancient university there.

In his first year in medical school del Río was fortunate in having the science of histology taught to him by Professor López García, who had trained under the French histologist Ranvier. After graduation del Río returned to Portillo to practise medicine among his townsmen, but within a few months he became convinced that he would be happier in laboratory work at the university. His former histology professor appointed him as his assistant, and del Río began work on his Doctorate of Medicine thesis entitled "Alterations of the nervous system in brain tumors." The results of this work were incorporated in his first publication, which appeared in the local medical journal *La Clínica Castellana* in 1909.

The next chapter in del Río's life began when he went to Madrid to see the already famous Ramón y Cajal, bearing a letter of introduction from a mutual friend, Dr. Leonardo de la Peña, the Professor of Anatomy at Valladolid. Del Río's first contact with the hard-driving investigator with the rugged Aragonese peasant philosophy, was somewhat disconcerting. After spending a short time in the Department of Histology at the University of Madrid, del Río won a travelling fellowship and took up studies in Paris with the distinguished French master of histological technique, Prenant. His grounding in the French school of histology with Ranvier's pupil, López García, in Valladolid stood him in good stead now, and he became an accomplished investigator under Prenant, publishing his work on the ovary in

* Read at the Pío del Río-Hortega Memorial Meeting of the Montreal Neurological Society, October 31, 1945.

† Dr. Miguel Prados, neurohistologist and psychiatrist, began to work in the laboratory of del Río-Hortega in 1918 and was his first pupil in neurocytology. Dr. William C. Gibson went to work in Hortega's laboratory in 1936 and was there when civil war broke out. Dr. Gibson was at that time working for his Doctorate of Philosophy at Oxford, England.—Ed.

‡ The University of Valladolid was founded in 1346.

1913. He travelled extensively in Europe and studied the most modern experimental techniques in pathology in London and later in Berlin.

On returning to Madrid he found that scientific circles were being rejuvenated by one of Cajal's senior disciples, Nicolás Achúcarro, who had recently come home to Spain after postgraduate work in America and in Alzheimer's laboratory in Germany. The first problem which Achúcarro attacked with his new armamentarium was that of the changes in the nervous system in animals suffering from rabies. This led him directly to the problem of neuroglial reactions in this disease and hence to one of the central problems of neurohistology at that time, the need for a better neuroglial staining method. He developed the tannic acid and silver technique which bears his name today.

Cajal realized that Achúcarro was the man who could best develop the unexplored field of neuropathology with the existing metallic stains and with the fund of knowledge of the normal histology of the nervous system which had been largely built up in Spain. He, therefore, helped Achúcarro to set up a small laboratory of neuropathology in the Museum of Natural History. It was to this laboratory that del Río was attracted on his return, as much by Achúcarro's personality and vigor as by the opportunity for research in neuropathology. In this atmosphere of strict scientific discipline and stimulating discussion del Río passed some of the happiest years of his life. He never forgot Achúcarro's generosity to younger men embarking on a career of medical research and teaching.

As soon as Cajal was able to enlarge his "Laboratorio de Investigaciones Biológicas" he invited Achúcarro to bring the laboratory of neuropathology under the same roof, so that normal and pathological histology could be carried on side by side. Thus it was that del Río, as Achúcarro's pupil, finally joined Cajal's enlarged group in Madrid. His training had been more French than Spanish, and until coming to Achúcarro's laboratory his work had been done with aniline stains rather than metallic.

Nicolás Achúcarro died while still in his thirties, leaving a great hiatus in Madrid's scientific life. Del Río became his successor in the field of neuropathology and kept the neuropathological corner of Cajal's laboratory humming with work on the neuroglia. In his hands Achúcarro's tannic acid-silver method became a dozen different methods, so rapidly did he develop what he modestly called *variantes*. Students who suggested that he publish a set of "standard methods" were regarded with deep misgivings, for he believed that if a research man were to master the methods of his craft he would eventually come to have his own best *variantes* of a basic method. And, conversely, del Río felt that a man who would turn over the silver staining of research material to technicians was not to be counted among his pupils.

Del Río succeeded Achúcarro as Director of the Laboratory of Neuropathology and was also made Pathologist to the General Hospital in Madrid.

In the latter capacity he devoted his mornings to the examination of

surgical and postmortem specimens. He set up a Pathological Museum at the hospital and later put it in the capable hands of his pupil, Dr. Pérez Lista, on his return from postgraduate training in Munich.

With the morning's work completed, del Río would go home for lunch and then directly to his *tertulia* at the coffee-house, where friends from various callings gathered for their little "parliament of culture." There the most divergent views were disputed by artists, scientists, writers, musicians and politicians in this typically Spanish institution. Usually Don Pío merely listened, and spent most of his time elaborating the notes or drawings that he had made during the morning's work in the laboratory. He was a man of deep conviction and only when some basic question was being discussed would he express himself. When he did, however, it was with such finality that the *tertulia* would sit up with a start. Promptly at three o'clock del Río would pick up his drawings and go off to his laboratory where he would work late into the evening.

It was natural that while working with Achúcarro, del Río's interest should have been stirred in the neuroglia. Achúcarro's last paper dealt with the "gliotectonics" of the various animal species and outlined an hypothesis as to the possible endocrine role of the astrocytes. No such theoretical function was advanced for the remaining neuroglial elements because they were, at that time, merely a conglomerate known as "the third element." (The neurons and astrocytes were the first and second "elements," respectively.) It was into this confused field of neurohistology that del Río's consuming curiosity and loyalty to Achúcarro took him.

The histological analysis of the "third element" had, for some time, occupied Cajal in Spain and Robertson in Scotland, the latter worker having used the term "mesoglia" to describe it. Both conjectured that a mesodermal element might exist within the nervous system, but they had never succeeded in staining completely the type cells of which they wrote.

Histologists everywhere were, therefore, surprised when the young del Río discovered, with his new silver carbonate stain, that the "third element" contained two distinct cell types. His first discovery was the "microglia," the spidery little cells designated by Spatz as "Hortega cells." A few days later a subtle variant of the same method brought the "oligodendroglia" to light, the name having been coined to emphasize the scarcity of their protoplasmic processes, in contradistinction to the astrocytes.

As del Río celebrated with his friends in the Plaza de Santa Ana, his mind raced ahead to the new problems which his silver carbonate technique had suddenly brought within range. He wanted to track down the origin of the delicate microglia and their function. The satellite oligodendroglia seemed to be standing guard around the neurons and their processes, but their function must be more than that, he thought to himself.

In the days, even the years, that followed, he pursued these cells until he knew their innermost secrets. He worked feverishly to extract answers to the problems that had been posed for years concerning phagocytosis in the

central nervous system, and the nature of the tumors arising from non-nervous tissues of the brain. His pupils joined in the unremitting search and in the process became some of the outstanding neuropathologists of the world.

As del Río piled discovery upon discovery, a strange coldness crept into Cajal's attitude in the laboratory. He believed that the cells "discovered" by his young disciple had already been described by Robertson, and Cajal sought to minimize the value and originality of del Río's work. In this Cajal was wholly in error, as an examination of Robertson's slides and papers subsequently proved.

Finally del Río withdrew from his master's laboratory to a little corner in the General Hospital, and with him moved the "centre of gravity" of the Spanish school of neurohistology.

With the help of the *Junta* in charge of postgraduate studies and research in Spain, del Río set up a Laboratory of Normal and Pathological Histology at the Student's Residence in the Pinar district of Madrid. This little laboratory belied its imposing name, for the space was limited and pathological material was hard to obtain. Though the material difficulties were legion, del Río's pupils stayed loyally on. Working *codo con codo* with him, they became intensely proud of their little laboratory. Their only longing was for access to Cajal's well-stocked library. The work of the new laboratory was published by the neighboring Museum of Natural History in its *Boletin* and its *Memorias*.

Much of the success of del Río's new laboratory at Pinar was due to the warm-hearted generosity of Dr. Juan Negrín, who was Professor of Physiology at the University of Madrid, and later became the wartime Premier of the Spanish Republic. Negrín gave del Río half of the very limited space that he had and all the chemicals and apparatus that were needed. Negrín was interested in neurophysiology and his ideas on decerebrate rigidity or cerebral metabolism would be as keenly debated in del Río's laboratory as in his own. Negrín's eldest son became a pupil of del Río. In fact once del Río started work at Pinar the students vied with each other to train under him. The *Residencia* became conscious of a new ferment in the laboratory building and many non-medical students would look in to see the now famous "Don Pío" at work. Among the best known of these was Frederico García Lorca, the poet, who joined del Río's *tertulia* and remained one of its luminaries until he was executed by a fascist band in Granada in 1936.

By 1922 del Río's first pupils were taking his histological techniques abroad with them during their postgraduate clinical training. In London, Sir Frederick Mott and Da Fano were the first to adopt them in their laboratories.

In Germany, Spatz and Metz published the earliest confirmation of del Río's discoveries. At the same time foreign students came to Madrid to learn at first hand the staining methods that had so completely altered the field of neuropathology. Wilder Penfield was the first to arrive, and through

his writings del Río's work became better known to the English-speaking world. A close friendship developed between them which grew richer with the years. On returning to America, Penfield, with his associate William Cone, adapted del Río's methods to systematic neuropathology. Their early work in the Laboratory of Neurocytology at the Presbyterian Hospital in New York did much to equate the two rapidly developing fields of neuropathology and neurosurgery. Kubie and Stevenson were among the earliest Americans to study with del Río. In a few years his trans-Atlantic pupils almost outnumbered his Spanish disciples.

From Buenos Aires came an invitation by the Institute of Spanish Culture asking del Río to give a series of lectures on his work. He went out to Argentina on the condition that he should have a laboratory at his disposal. On his arrival he set up a miniature school in the laboratory and in a few weeks he was able to train a large group of pathologists in his staining methods. Little did he realize that this same group of devoted pupils would some day offer him a refuge and a laboratory in exile.

Back in Madrid del Río developed a remarkable group of Spanish disciples at the *Residencia*, many of whom later became well known internationally. The early group included Jiménez de Asúa, López Enriquez, I. A. Costero, the Gallegos, Ortiz Picón, Luis Sacristán, Vázquez López, Alvarez Cascos, Solís and Escardo. In addition to these postgraduate students, del Río trained a large group of undergraduates from the *Residencia* in the elements of normal histology. It was not long until his students began to call him, with affection and respect, "Don Pío," signifying that he was their master in the science and art of histology.

Once del Río had cleared away some of the major mysteries in the normal histology of the nervous system, he turned to the tumors and malformations to which it is heir. Though his laboratory lacked direct access to pathological material, the deficiency was made good by Dr. José Goyanes, then Director of the Cancer Institute of Madrid. In a short time the importance of del Río's work became so apparent to the Institute that he was made Chief of its Department of Experimental Research.

The increasing demands on his time, and the complexity of his experimental work, gradually forced Don Pío to depend on photomicrography for the routine illustration of his specimens. But he never lost his love of painting beautiful microscopic fields. He often remarked on the cunning with which cancer masqueraded under his microscope as an enchanting picture, and some of his finest paintings were of cancer. At home Don Pío turned to oils and painted many pleasing scenes, though few of his friends knew he was their author. He was a devotee of the Greek refugee Theotokopoulos, whom the people of Toledo called "El Greco." Each student who studied in Don Pío's laboratory came to share in the admiration that he felt for Toledo with its matchless paintings, its architecture and its tradition. Sunday found the mob on its way to the Plaza de Toros in Madrid but it found Don Pío and his students in Toledo studying El Greco's art.

In 1932 the Cancer Institute in Madrid was reorganized and a new building was erected at the University City on the outskirts of Madrid. Del Río was appointed as Director and he set about organizing a diagnostic and therapeutic service for the whole of Spain. He at once proceeded to publish the *Archivos Españoles de Oncología* in which the research work of the Institute was reported. In 1933, on the occasion of the International Cancer Congress in Madrid, del Río published a beautifully illustrated monograph, *Tumores del Sistema Nervioso.* This comprehensive volume summarized his contributions to systematic neuropathology, but it does not contain any of his work on normal histology or experimental cytology. In 1937, while the Luftwaffe was destroying the University City in Madrid, a German scientific publisher asked permission to publish a translation of the tumor book. Del Río replied that as a Spanish scientist and democrat he would be humiliated to have anything published in Hitler Germany.

By 1935 the laboratories at the Cancer Institute and in Professor Negrín's little building at Pinar were too small to accommodate the steady stream of pupils from abroad. The *Junta* and the *Residencia* therefore asked del Río to design a building suitable for his research and teaching. The building that they constructed dwarfed the little Pinar Laboratory and was a model in brightness and simplicity. After fifteen years of makeshift Don Pío was able to show as fine a building as there was in Europe to students who came from abroad to "The Laboratory of Normal and Pathological Histology."

The established routine became:—mornings at the Cancer Institute and afternoons and evenings at Pinar. The demands on Don Pío's time were heavy at the Cancer Institute so that his instruction was limited to "rounds" at 9 a.m. and 12 a.m., but at Pinar he was almost constantly with his pupils. He would move from cubicle to cubicle, encouraging the novitiates as they struggled with the elusive "oligo" or the fickle microglia. After looking at the slides so laboriously made, Don Pío would pour fresh solutions into the glass *pocillos*, adjust the height of the alcohol flame, take off his glasses and begin blowing on the frozen sections floating in a Petri dish to separate them without damage. To watch him carry the feathery little sections through the staining processes to the final dropping on of the coverslip was an unforgettable experience. The charm of his teaching lay in his modesty and in his ability to see himself, in retrospect, as a student who once struggled with the refractory tissues of the nervous system. He never forgot with what patience and encouragement Achúcarro had initiated him into silver staining.

The spirit that dominated del Río's laboratory was not one of routines and stereotyped drills in technique. Each block of tissue was "sized-up" as to its bulk, the best method of embedding it, the fixation time and the stains to be used. There was no mass production psychology in the training. Del Río would sit down with each student in turn, and he spent most time with those furthest from their goal. If someone came from abroad with a new problem, it immediately became del Río's problem too, for he delighted in the unusual and in the things that challenged his ingenuity.

In retrospect it is easy to see the increasing emphasis that he placed on techniques which could wring the greatest amount of information out of the smallest piece of tissue. He evolved gelatin embedding methods for friable tumors and for small objects, such as ganglia, without altering the silver staining procedures whatever. Frozen sections came to replace the paraffin and celloidin embedding techniques to such an extent that del Río began to cut sections of whole brains on his large freezing microtome. It was a lesson in patience to watch him guide these huge sections through his double impregnation technique and finally mount them on a slide the size of a quarto volume.

When Ramón y Cajal died in 1934 it was expected that del Río would succeed him in his seat in the Academy of Medicine in Madrid. However, the Academy, acting as the French Academy had done in Pasteur's case, barred Spain's most distinguished biologist. The outcry was immediate and vehement by the younger scientists, artists and writers. Under the leadership of Gonzalo Lafora, one of Cajal's pupils and sub-director of the Institute Cajal, a great testimonial meeting was held in Madrid, at which del Río's contributions to science and to Spain were movingly recognized. It was symbolic of the rift developing in Spanish life between those who stood for progress and those who wanted to remain in the past.

One of the signs of progress in Spain was the conversion of the former royal palace at Santander on the north coast into an international summer university to which students from abroad came to perfect themselves in the Spanish language, and Spaniards came to study the culture of the other countries of Europe. Every summer Don Pío would come to Santander to escape the heat of Madrid and to work in this congenial atmosphere. His laboratory was housed in the beautiful Casa de Salud which had been built by his friend the Marquis of Valdecilla. It was Don Pío's failure to arrive at his laboratory from Madrid on the day in July 1936 that he had specified which led the Summer University to suspect that trouble was afoot in the Capital.

Providentially for the Spanish Republic the armed insurrection prepared by a group of Spanish generals and their allies abroad broke out before the expected bombers arrived from Italy. The government's initial advantage, however, was soon overshadowed by the might of the Luftwaffe which undertook non-stop raids from bases as far distant as Hamburg. One of the first casualties in the war was del Río's beautiful new Cancer Institute on the edge of the University City in Madrid. The Moors and Italians made a shambles of the newly constructed medical school at the base of a bluff dominated by the large Hospital Clínico.

Río-Hortega's pupils led a group of soldiers to the ruined Cancer Institute under cover of darkness and succeeded in saving its supply of radium from loss or destruction. Don Pío carried it with him to Valencia and handed it over to the government. The younger members of his research group went off to the front, while the older ones organized a neurosurgical unit in Madrid.

The first of his pupils to be killed was the young Gallego, the son of del Río's early associate Professor Abelardo Gallego. Young Gallego had been Don Pío's chief assistant at the Cancer Institute and kept the large collection of slides and records. His death in the campaign against the Italians at Brunete was one of the causes of Don Pío's rapid aging during the war.

Even though the Junkers pulverized Carabanchel where Cajal had his week-end cottage, and dropped incendiaries on the Prado Art Gallery and the neighboring Instituto Cajal, del Río stayed on in Madrid, giving his pupils and their hospital all the support of his respected position in the community. The entreaties of foreign universities and research institutes asking him to leave Madrid fell on deaf ears. Finally, the government, which had moved to Valencia, sent for him, and prevailed upon him to stay there in La Casa de la Cultura which had been set up as a refuge for Spain's older men of letters. His departure from Madrid was a heart-rending experience for his pupils who saw him off on a crowded bus for Valencia. His pockets were crammed with microscope lenses and his luggage was laden with his priceless collection of slides. Most of all, his heart was broken at the sight of mile after mile of refugees streaming south from Madrid, a scene of suffering soon to be repeated around the world.

No sooner had del Río settled down to work at the Histological Laboratory of the University of Valencia than the military situation deteriorated still further. The Prime Minister, Dr. Juan Negrín, strongly urged del Río to accept an invitation to work in Dr. Clovis Vincent's Laboratory at L'Hôpital de la Pitié in Paris. Professor Vincent's clinic had long been del Río's source of supply for tumors of the nervous system for histological study. Believing that the move to Paris would enable him to complete an enlarged second edition of his book, del Río set out for France. Again the roads were filled with refugees driven by the relentless Luftwaffe.

While in Paris del Río was invited by the Nuffield Foundation at Oxford to organize a laboratory of neuropathology in the recently established clinic of Hugh Cairns, the Nuffield Professor of Surgery. In the autumn of 1937, Don Pío, accompanied by his sister and niece and by his life-long friend Nicolás Gómez del Moral, came to Oxford for what was to be a sojourn of three years.

Don Pío drew up plans for the laboratory along the lines of his recently destroyed unit at the Cancer Institute in Madrid. To see it by night one might imagine oneself back in Madrid, but by day there was no mistaking the grey sky overhead and the short days of England. Cajal's dictum, "Grey matter flourishes under grey skies" was fully borne out in Oxford, much to del Río's delight. The multiplicity of cultural societies in the University was a constant source of wonder to Don Pío. The frequent degree-granting ceremonies with their orations in Latin appealed strongly to his love of tradition. He was intensely proud of the honorary degree of Doctor of Science which Oxford conferred on him, especially since a similar degree was conferred on Harvey Cushing in 1938.

The quiet of the laboratory at Oxford was in marked contrast to the bombing of Madrid, and Don Pío often fell into a reflective mood, humming or singing Spanish folk songs as he worked. Sometimes it was a lilting melody, but oftener it was a sad little tune. He knew the songs and dialects that characterized each region of Spain and he loved to teach his students the rhyming proverbs which he had learned in Portillo as a boy. He learned the American Negro song "Water Boy" from hearing it in the laboratory. Its sadness haunted him and he set up a copy of it on his bench so that he could hum it while waiting for sections to stain.

In November 1938, del Río gave two public lectures at Oxford on "The microglia" and "The oligodendroglia" in which his work on these cells was summarized.

In commenting editorially on these lectures the *Lancet* said: "A great service has been done by Wilder Penfield in making the English-speaking people more familiar with the recent Spanish work, but it is even better that we are able to publish a lecture given in this country by Hortega himself." After recalling the incredulity with which the discoveries of Cajal and del Río were first greeted, the editorial concludes: "The latest confirmation of Hortega's conception comes from Kershman, a pupil of Penfield's at Montreal, who in a masterly study of the first comprehensive series of human embryonic and foetal material to be so investigated has mapped out the various sites at which the microglial cells make their entry into the nervous system."

The outbreak of war in 1939 brought del Río increasing responsibilities and sadness, for many of his compatriots were living as exiles in France. He became a familiar figure at the telegraph office in Oxford as he sent cables to Mexico and South America seeking asylum for his former colleagues and pupils.

It was in the midst of these anxious days that he became aware of the illness from which he was eventually to die. A diagnosis of incurable cancer was made in London in 1940. Don Pío then decided to make his will and left his library and priceless collection of slides to the Casa de Salud de Valdecilla, in Santander. The witnesses were the two men who had been his counsellors and supporters in Madrid, the Secretary of the Junta para Ampliación de Estudios, and the Warden of the Residencia de Estudiantes at Pinar. He could not return to Spain and yet he wanted to live his remaining days in a Spanish-speaking country. He, therefore, accepted an oft-repeated invitation of the Spanish Institution of Culture in Buenos Aires and set out with the faithful Nicolás Gómez for the Argentine.

He was warmly welcomed by such leaders of scientific thought in Argentina as Professor Houssay, but already their academic freedom was imperilled by the fascist military clique which dominated that country. However, del Río was not to be coerced or dictated to by such tyrants, and he set up a modest laboratory in which to train another group of "disciples." Two of these, Dr. Polak and Dr. Prado, were his closest collaborators and published several papers with him in his new *Archivos de la Sociedad Argen-*

tina de Anatomía Normal y Patológica, the last one appearing in 1944. He threw himself into his teaching as he had into his research in the early days of the microglial discoveries, for he realized that time was running out. When he could no longer go to the laboratory he turned to writing a book on histological technique at home. Finally, when he could not even hold a pen, he dictated the text. One of his greatest hopes was that he could finish the life of Cajal which he had been writing for years.

A few months before his death Don Pío wrote to one of his students: "I am now working harder than ever before in my life, though I will never know what the results will be." With such a spirit it is little wonder that Don Pío declined General Franco's invitation to return to Spain now that an early end was inevitable. He quietly reminded the dictator of the protest the Spanish Rebels had made to the British Government when Oxford University honored Don Pío with its highest degree. To the end there was no compromise between Spain's greatest scientist and the barbaric Falangist forces of that country.

Pío del Río-Hortega died in the early morning of June 1, 1945. With the stilling of his generous heart and his active mind, the world lost one of its rarest spirits, and Spain its most cultured scientist. As Cajal had typified progress to an earlier generation, so del Río epitomized the incorruptible fibre of modern Spanish thought. His simple Castilian faith in the essential dignity and the ultimate value of scientific research he carried throughout his life. For him, freedom of thought and intellectual self-respect were paramount.

The truth of Cajal's dictum "For scientific work the means are almost nothing and the man is almost everything," was borne out completely by del Río's life. He was loyal to his teachers, his pupils, his friends, his native land, to science and freedom.

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FACTORS CAUSING MASSIVE SPASM FOLLOWING TRANSECTION OF THE CORD IN MAN*

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SINCE the first World War the idea has become prevalent that when the spinal cord has been completely transected in man, after a brief period during which the reflexes are abolished, there develops, inevitably, a chronic state of heightened reflex activity in the isolated cord, leading gradually to mass reflex spasms, which are considered to be the result of "release" of the isolated portion of the cord from the influence of the brain.

These current views were foreshadowed by those of Romberg,[14] Charcot,[5] Gowers,[7] Riddoch,[13] and others. Romberg[14] wrote:

> When motility and cerebral sensibility are entirely lost, spinal or unconscious sensibility manifests itself by reflex action . . . . In spinal injuries some time elapses before these phenomena are manifested, a circumstance probably dependent upon the concussion of the organ . . . . The antagonist muscles assume a state of permanent tension . . . . The contraction of the flexors is occasionally so considerable, that the heel touches the nates.

Charcot[5] in his lectures on spinal injury taught that:

> In the first degree, simple paresis is observed with flaccidity of the limbs . . . . At the end of some days or of some weeks, later in certain cases, sooner in others, there supervene, in the paralyzed members, jerks, cramps, a temporary rigidity of the muscular mass,—so many symptoms attaching also to lesion of the lateral columns, but which already indicate that a source of excitation has taken up its abode in the fascicles . . . . Lastly, supervenes permanent contracture of the limbs . . . . It is the rule that this contracture shall first, for some time, maintain the paralyzed limbs in a posture of forced extension, but sooner or later generally speaking, a posture of forced flexion supervenes . . . . In this phase of the disease, under the combined influence of *the suppression of the moderator influence of the brain* . . . the reflex properties of the inferior segment of the cord are augmented. Then, we see the paralyzed members rise and become convulsed at the least touch, or when the patient micturates or goes to stool.

In 1886, Gowers[7] said:

> An acute lesion in any part of the cord may cause an initial loss of reflex action in the part below, but if the lesion is above the lumbar enlargement, reflex action returns in the course of a few hours. Frequently, there is no initial depression. Subsequently, the reflex action becomes excessive, that from the skin rapidly, that from the muscles more slowly. Ultimately, each attains a high degree of exaltation. . . . Cases of superficial reflex action indicate withdrawal of the cerebral controlling influence of the reflex centers.

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Although these views were briefly challenged by Bastian[1,2] and others,[3,4,6,11,12,15] they continued to represent prevailing opinion.

In 1917 Riddoch[13] published an important paper, which fortified the ideas previously expressed by Romberg, Charcot and Gowers, and has largely influenced all subsequent thought on this subject. Riddoch reported in great detail his observations upon five patients in whom complete transection of the spinal cord was verified at operation, four of whom lived for many months after injury. Each of these four patients, after passing through an initial period of so-called "spinal shock," eventually developed massive reflex movements of the lower extremities, which Riddoch believed to be the true chronic state of reflex activity in the human following transection of the spinal cord. Although he carefully avoided ascribing these mass reflexes

TABLE 1

Verified transections without mass spasms

Case	Level	Patient	Spasm	Interval Since Injury
1	C-7	"J"	0	6 mos.
2	T-4	"DK"	0	6 mos.
3	T-6	"S"	0	9 mos.
4	T-6	"JK"	0	9 mos.
5	T-10	"M"	0	9 mos.

Verified transections with mass spasms

Case	Level	Patient	Spasm	Interval Between Injury and Onset of Spasms
6	T-2	"W"	XXXX	1 mo.
7	T-3	"J"	XXXX	3 mos.
8	T-4	"R"	XXX	5 mos.
9	T-5	"McM"	XXXX	2 mos.
10	T-6	"Ba"	XXXX	1 mo.
11	T-6	"N"	XXXX	2 mos.
12	T-8	"Br"	XXXX	3 mos.

to the "release" of the isolated segments of the cord from the influence of the brain, there is no doubt that that is the general interpretation that was placed upon Riddoch's observations by others and is the view most widely held today.

In our experience, on the contrary, the chronic reflex state of man following transection of the spinal cord is highly *variable*. Of 12 patients with verified transection at mid and upper thoracic levels, studied by us, 7 developed mass involuntary spasms, while 5 did not. Furthermore, among the 7 cases in which spasms did develop, there was great variation in the interval of time elapsing between the transection of the cord and the onset of the spasms (Table 1).

These facts challenge the prevailing concept that "release" of the cord from the influence of the brain is the *sole* mechanism responsible for the

development of mass spasm following transection of the cord; and suggest that, in addition to "release," which is common to all this group, there were in those cases in which spasm developed, added factors not heretofore described that contributed to their occurrence. It was felt that these factors were essentially pathologic in nature and inherent in the lesion.

SURGICAL OBSERVATIONS

A. *Excision of Extrinsic and Intrinsic Scar Tissue from the Stumps of the Previously Severed Spinal Cord* (5 cases). The considerations above described led, in 5 patients suffering mass spasms, to surgical exploration of the lesions.

In each case the spinal cord was found completely divided, with the proximal and distal stumps connected by a strand of fibrous tissue. The first 1–2 cm. of each stump were grossly distorted and firmly anchored to the dura by massive adhesions. This portion of the cord was unresponsive to both mechanical and electrical stimulation. Immediately beyond this area, however, in the distal stump, there was a "transition zone" in which the cord began to assume a normal appearance; and here, minimal mechanical and electrical stimuli initiated mass contractions which were greater than those obtained with the same stimuli applied to more distal segments. From these observations it would appear that the area of anatomic transition represents a region of hyperexcitability or "trigger zone." Within this zone the maximal responses were obtained from stimulation of the dorsal columns, while lesser responses were elicitated from the ventro-lateral portion of the cord and the dorsal roots. Stimulation of the ventral roots produced only a segmental response.

In 4 patients both the distal and proximal stumps of the cord were released from adhesions binding them to the dura, and excised. In one case the proximal stump, alone, was excised. Microscopic examination of these stumps revealed in each instance marked distortion of the normal architecture and extensive degeneration and gliosis.

Following operation there was in each instance an alleviation of spasms ranging from 20 to 90 per cent. This improvement, however, was only temporary, and after varying intervals of time, ranging from 2 to 10 weeks, there began a gradual increase in the spasms.

Case 1—"*B*". On 7 October 1944, this patient received a gunshot wound of the spine at the level of T-4 resulting in immediate complete sensory loss and flaccid motor paralysis below his wound. One month after injury, involuntary spontaneous reflex spasms began, increasing in severity so that they had become almost constant and quite violent, forcing the patient to assume, while in bed, a position in which the thighs were acutely flexed on the abdomen and the legs acutely flexed on the thighs. Even with forceful and sustained traction on the ankles, the legs could be straightened only a few degrees due to the violence of the flexor spasms. There was no evidence of any return of motor, sensory or bladder function.

On 25 September 1945, laminectomy was performed under local anesthesia at the level of his injury. The spinal cord had been divided by his original wound, leaving only a tough, gritty strand of scar. This was glued to the dura by dense adhesions, as were the distal and proximal stumps of the cord itself. The tough fibrous scar, after tests had showed it conducted no nerve impulses, was excised. Adhesions around the diseased ends of the cord were freed.

At the lower end of the scar, in obviously diseased spinal cord, was a trigger zone which was removed with the scar. Microscopic section of this scar showed that no nerve fiber of any kind ran through it. Section through the diseased trigger zone at the lower end of the scar showed considerable loss of myelin, internal scarring and disorganization.

Following this operation, the patient, though not cured of his spasm, was definitely better. On 30 October, 5 weeks after the laminectomy, he was observed continuously for 4 hours, during which time he had only 3 very mild spontaneous spasms of his legs. At that time, it was no longer possible to elicit mass reflexes by cutaneous irritation and it was difficult to initiate them even with strong proprioceptive stimulation. In early November, he traveled to a nearby city to see a football game and in December he made a long journey to his home on a furlough. Since then, however, follow-up reports indicate that there has begun a gradual increase in the frequency and severity of spasms.

Case 2—"*R.*" In February, 1944, this officer received a penetrating wound of his spine at the level of T-4 resulting immediately in complete sensory loss and motor paralysis below that level, which have persisted to date without any signs of recovery. Five months after injury, spontaneous mass reflex spasms of his legs began. These spasms increased in frequency and severity. They would occur after any cutaneous or proprioceptive stimulus. They were frequently initiated by a change of position in bed. They often lasted for 20–30 minutes and would be so severe that they would audibly "knock the wind" out of him and leave him exhausted.

On 28 September 1945, under novocain anesthesia, laminectomy was performed. The findings and the procedure were similar to those described in Case 1, but the excision of cicatrix and scarified cord was less radical and complete than in some of the later cases.

Following operation, there was slight but definite improvement. There were no longer reflex spasms following cutaneous stimulation of the legs, and on many occasions it was not possible to elicit spasms by any form of stimulus. On 10 October 1945, it was estimated that his spasms were only about 50 per cent as severe as they had been before his operation. On 12 October 1945, this patient set out with his mother in a car for his home in California. He has not been seen by the writers since that time, but follow-up reports have indicated a gradual return of the spasms of the legs, although not to their original severity.

Case 3—"*N.*" In November, 1944, this patient received a gunshot wound of his spine at the level of the 6th thoracic segment which resulted in immediate and total loss of sensation and power below that level, which has shown, to date, no signs of recovery. A few months later, reflex spasms of his legs began.

On 24 September, under novocain anesthesia, his spine was explored at the site of his wound. At that time, his legs were in such a state of spasm that both hip and knee joints were almost continuously flexed to right angles. He had to be forcibly strapped to the table and even then, during the greater part of the operation, his pelvis was well off the table.

The findings were similar to those described in Case 1. Scar tissue and the cicatrized upper and lower stumps of the previously severed cord were excised. On 28 October, 5 weeks after operation, he had only a few spontaneous spasms. He no longer has reflex spasm on cutaneous stimulation of his extremities, although squeezing his quadriceps femoris muscle still causes marked spasm of the muscle. Girdle pains are very much reduced in both frequency and severity. They no longer cause nausea or vomiting. The patient has gained weight. Improvement is estimated at 40–50 per cent.

Case 4—"*Br.*" On 21 September 1944, this patient received a gunshot wound of his spine with immediate loss of all sensation and motor power below the level of the 5th thoracic segment, which has persisted to date. Several months later spontaneous massive reflex spasms began which became increasingly worse.

On 9 October 1945, under novocain anesthesia, laminectomy was performed at the site of the injury and the lesion exposed. The cord was reduced for about 2 cm. to a hard, gritty band of tissue having a cross section about $\frac{1}{2}$ that of the normal cord, tightly bound to

dura with dense adhesions. These adhesions were divided, and the fibrous band and adjacent cord freed of adhesions. Nothing further was done at this session.

Following this first operation the patient had violent spasms of the abdominal muscles and also of the visceral musculature. These latter were associated with nausea, vomiting and simultaneous fecal discharge from the rectum. These continued for several days.

On 12 October 1945, the laminectomy wound was reopened under nitrous oxide-oxygen-ether anesthesia. At this time, the lightest possible touch of the distal stump of the cord with the tip of the forceps, at a point about 1 cm. below the hard fibrous band, was sufficient to set off massive reflex spasms of the legs. The slightest traction on cord or roots at this point produced similar results. Maximum spasms of legs occurred when touch was applied over the dorsal columns.

Electrical stimulation of the hard fibrous band gave no responses at all, even with very strong currents. Stimulation of the dorsal surface of the cord 1 cm. lower gave quick bilateral flexor mass spasms of the legs with equally quick relaxation; while stimulation of the dorso-lateral and ventro-lateral surfaces of the cord produced much slower and less violent contractions and relaxations. There were no nerve elements in the scar on microscopic examination.

The hard gritty band of scar tissue was excised, together with about 3 cm. of the distal stump. In this was found a fair-sized syrinx occupying the ventral half of the gray matter and extending from the site of the lesion well beyond the lower limits of the excision and the surgical exposure.

Immediately after the operation, the patient had continuous clonic spasms of his legs which lasted for 12 hours, thus proving the absence of spinal shock following secondary transection in this case. After that, spontaneous reflex spasms gradually subsided. On 28 October they were less than half as severe as they had been before operation. Cutaneous stimulation no longer set off spontaneous spasms, though these could still be induced by proprioceptive stimuli. The legs no longer had to be tied down when the patient was in a wheelchair, which had been the case prior to operation. However, in the latter part of November, there was a gradual increase in the frequency and severity of the spasms, although they never attained their preoperative frequency and severity.

Case 5—"*J.*" This young man sustained a gunshot wound of the spine at the level of the 6th thoracic vertebra in November, 1944. Immediately following this, he had complete motor and sensory loss below the level of his lesion which never showed any sign of recovery. A few months later, he began to have massive spasms of his legs, which gradually increased until his legs had to be tied down even while he was in bed. At the same time, he had severe girdle-like pains at the upper level of his anesthetic area. This pain and the spasms became so bad that the patient could not eat or sleep and death seemed imminent from undernourishment and exhaustion.

On 18 August 1945, under nitrous oxide, oxygen and ether anesthesia, laminectomy was performed primarily for relief of pain through rhizotomy. The cord at the site of injury was found to be shrunken, hard and partly ossified. Both it and the spinal roots were involved in the densest sort of scar tissue. It was found impossible to dissect out the roots in such a way that they could be sectioned between scar and cord. Accordingly, a block resection of about 3 cm. including degenerated cord of the proximal stump was carried out. The distal stump was not resected.

After operation, the patient's pain was entirely gone. He has had a few occasional small spasms of his toes, only. He has gained 15 lbs. in weight and is up in a chair every day.

In contrast to the 5 cases of spasm above described, one additional patient without spasms was explored at the site of earlier traumatic transection for relief of root pain. Essentially the same pathologic picture was found as in the preceding cases and similar effects were obtained upon mechanical and electrical stimulation. After resection of the desired nerve roots, the

cord was freed of adhesions to the dura but none of it was resected. Six weeks later, spasms appeared in this patient for the first time since he had been wounded 9 months prior to the operation. In this patient, once again, an operative procedure upon the distal stump of the cord had apparently affected the reflex activity of the isolated segments.

Case 6—"*J.K.*" In February, 1945, this patient received a shrapnel wound of the spine resulting in complete paralysis below the level of the 7th thoracic segment, associated with severe root pain at the level of the lesion.

On 15 November 1945 laminectomy was performed at the site of the lesion for the relief of the root pain. Essentially the same pathologic picture was found as in the preceding cases, and similar effects were obtained following mechanical and electrical stimulation after section of the desired nerve roots. The cord was then freed of adhesions to the dura, but none of it was resected.

Six weeks after this operation, mass spasms appeared in this patient for the first time since he was wounded, and rapidly assumed major proportions; and it would appear that the surgical manipulation upon the distal stump of the cord had caused a persisting increase in the reflex activity of the isolated segments.

The 6 cases just reported indicate that operative procedures carried out upon the distal stump of a transected cord are capable of affecting the mass reflex activity of the isolated segments.

B. *Surgical Section of Dorsal Columns Below the Level of the Spinal Lesion* (2 cases). In the preceding cases both mechanical and electrical stimuli had produced maximum contractions when applied to the dorsal columns, thus suggesting that these columns, functioning in an antidromic or descending direction, were important channels for the dispersion of stimuli throughout the isolated portion of the cord. For this reason, in two patients with high thoracic cord transection, suffering spasms, the dorsal columns were divided below the level of injury. Section was carried out in each patient, at two separate levels, at the 10th thoracic segment with the idea of isolating the lumbosacral segments from abnormal impulses arising within the stump, and again at the 12th thoracic segment in an effort to reduce the dissemination of afferent impulses reaching the caudal segments of the cord from the periphery.

Following this procedure, there was striking relief from spasms in one patient which lasted three months. In the second patient, improvement was only transitory.

Case 7—"*W.*" In July, 1944, this patient was wounded in the spine resulting in an immediate and lasting loss of sensory perception and motor power below the level of the 4th thoracic segment. Spasms began in the legs and abdominal muscles 1 month later, and became progressively worse, until they were quite severe and recurred spontaneously almost constantly. They could be initiated if the foot were stroked, the legs passively moved, or the patient ingested food.

On 14 October 1945, under novocain anesthesia, laminectomy was performed from T-8 to T-12 laminae, inclusive. The arachnoid membrane was moderately thickened, suggesting a previous widespread inflammatory reaction. The lightest possible touch applied with the tip of the forceps to the dorsal columns caused massive spasms of the lower extremities. Similarly, any mechanical stimulus applied to the dorsal roots would set off massive spasms.

Electrical stimulation produced maximum effects when applied to the dorsal columns, next when applied to dorsal roots. In each instance, the spasms were quick and the relaxations were quick. Stimulation of the ventral part of the cord gave sluggish, slow contractions of the legs with equally slow relaxations; while stimulation of the ventral roots gave the poorest mass response of all. By using weak stimuli, it was possible to obtain responses on one side of the body only. The dorsal columns were sectioned bilaterally at the level of the 10th and 12th thoracic vertebrae.

Immediately after operation, there was complete abolition of spontaneous massive spasms of the lower extremities; nor could spasms be induced by any form of cutaneous or proprioceptive stimulus.

At the time of writing this report, on 4 November, cutaneous stimuli failed to elicit spasms and the strongest possible proprioceptive stimuli produced only the very slightest movement of withdrawal; while no spontaneous mass movements at all have occurred since operation.

These cases demonstrate that the severity of mass reflex spasms of the lower extremities following transection of the spinal cord in man, may be definitely reduced by surgical section of the dorsal columns below the level of the lesion.

SEGMENTAL REFLEXES

The tendon reflexes in patients with transected cords and mass spasms were, prior to surgical resection, generally exaggerated. As regards the Babinski sign, there was up-going of the hallux in some cases, down-going in others, and no response to plantar stimulation in others. After surgical procedures on the distal stump of the divided cords there was marked reduction of tendon reflexes in 4 cases and alteration in the responses of the toes to plantar stimulation.*

DISCUSSION

Clinical studies show great variation in both the segmental and mass reflex activity of the distal segments of the transected cord in man. No relation appears to exist between the level of the transection and the presence or absence of spasms, nor any constant time-interval between transection and the appearance of spasms.

These considerations make it clear that the occurrence of mass spasms following transection cannot be satisfactorily explained solely on the basis of the "release" of the isolated segments from the influence of the brain. That variations in reflex activity cannot be satisfactorily correlated with damage of particular conducting systems of the spinal cord has already been demonstrated in an experimental manner by Mettler,[9,10] while Lassek[8] has demonstrated a similar lack of correlation from a pathologic point of view in the human.

Surgical observations have revealed marked pathologic changes in and about the distal stump of the traumatically severed cord, which include, in

* A more detailed description of the segmental reflexes in these cases will appear in a subsequent communication.

addition to dense adhesions anchoring the stump to the dura, extensive degeneration and gliotic changes within the cord itself. It has also been established that this portion of the cord in the region of the injury has a lowered threshold for both mechanical and electrical stimuli; and that nervous impulses induced within this region, in response to stimuli, are readily transmitted to more distal segments by way of the dorsal columns. It has further been shown that surgical procedures that alter the anatomic condition of the stump, cause alteration of the reflex activity of the cord below that level. Finally, it has been demonstrated that striking amelioration of the spasms may be obtained by interrupting one of the main pathways (dorsal columns) for transmission of impulses from the stump to lower segments.

The *conclusion* is drawn from these observations that factors operating upon, or within, the isolated portion of the divided cord, especially at the level of the lesion, may play an important role in determining the occurrence, as well as the severity of mass spasms.

An "*irritative mechanism*" contributing to the development of massive spasms following transection of the cord in man is suggested by these observations. Constant or oft repeated traction upon the adherent stump of the distal segment, or the irritative effect of gliosis within the stump, may increase the irritability of this portion of the cord, and lower its threshold for afferent stimuli. Efferent impulses arising in this hyperexcitable zone are then transmitted to the more distal segments, chiefly, though possibly not entirely, by the dorsal columns in antidromic direction. It is fully recognized that "release" of the cord from the influence of the brain may play a primary role in *conditioning* the isolated segments for the development of spasms; but it is believed that the actual occurrence of spasms in any given case of a transected cord, is *determined* by factors operating upon or within the isolated segments, chiefly at the site of the lesion, and primarily irritative in nature.

SUMMARY

1. Evidence has been presented showing that massive involuntary spasms do not invariably follow transection of the spinal cord in man.

2. Studies indicate that when massive spasms do appear, their occurrence is determined not alone by "release" of the cord from the influence of the brain but by additional factors, among which *chronic irritation* of the cord due to extrinsic or intrinsic scarring of the distal stump at the site of the lesion appears to play an important role.

3. Electrical stimulation of the human spinal cord at the operating table in patients under local as well as general anesthesia is reported.

4. Relief from spasm, in varying degrees, has been obtained by the following surgical procedures:

(a) Liberation of the ends of severed cord from cicatrices binding them to the dura, and excision of degenerated tissue from the stumps of the cord (5 cases).

(b) Surgical section of the dorsal columns caudad to the lesion (1 case).

5. An "irritative mechanism" contributing to the occurrence of mass involuntary spasms in man following transection of the spinal cord has been proposed.

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DAMAGE TO PERIPHERAL NERVES BY HIGH VELOCITY MISSILES WITHOUT A DIRECT HIT*

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INTRODUCTION

IT HAS BEEN realized for many years that a high velocity bullet striking the body produces a wound many times larger than the bullet itself. The mechanism by which such extensive damage is done has been recently discussed by Black, Burns, and Zuckerman (1941),[2] Callender (1943),[4] Butler, Puckett, Harvey and McMillen (1945),[3] and Harvey, Butler, McMillen and Puckett (1945).[8] Reference to the early literature will be found in these papers.

Briefly, a missile of relatively small size entering soft tissues at high velocity produces a temporary cavity of large dimensions. Externally the temporary cavity can be recognized in spark shadowgraphs as a swelling (Black *et al.*, 1941).[2] Its exact contours have been demonstrated by roentgenograms made with an exposure of approximately one-millionth second (Harvey *et al.*, 1945;[8] Butler *et al.*, 1945[3]). The formation of this large cavity brings about a considerable displacement and tearing of tissues and offers a reasonable explanation of the abnormally large amount of damage produced by these small high velocity missiles.

It may be expected that, in addition to gross damage to tissues, which is produced by the direct impact of the missile, or by mechanical effects caused by the temporary cavity, damage less easily detected by ordinary observation may be caused to organized structures within the soft tissues. One type of structure, nerve, offers itself as a particularly convenient object to examine these possibilities of functional and anatomical damage in cases where the nerve was not struck by the missile directly. Nerve, like other components of soft tissue, should be subjected to marked displacement and deformation by the large temporary cavity that is produced by the passage of a high velocity missile.

That functional damage to nerves can result from the passage of a high velocity missile that did not strike the nerve directly has been reported by Seddon (1943)[11] and by Livingston *et al.* (1945).[9] However, the exact mechanism by which this damage to nerves is brought about does not seem to be well understood.

The object of the present investigation has been: (1) to demonstrate

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† On leave from Kansas State College.

visually that a nerve can be subjected to rapid displacement and deformation by a high velocity missile which does not strike the nerve directly and which produces no interruption in the gross anatomical continuity of the nerve and (2) that such displacement of the nerve can result in functional and anatomical damage to the nerve.

MATERIALS AND METHODS

A total of 21 living cats, under deep nembutal anaesthesia, were used in the present series of experiments. Missiles were fired into both thighs, giving a total of 42 shots.

The missiles used in the experiments were 4/32″ steel spheres, whose impact velocities were either approximately 3,200 ft/sec. or 4,100 ft/sec. The sciatic nerve was selected as the most suitable for study. The gun was aimed so that the sphere would traverse the tissues of the thigh at a point near the sciatic nerve, but every effort was made not to strike the nerve directly.

The sciatic nerve, in desired cases, was made radiopaque by the method recently described by French and Strain (1945).[6] A posterior incision was made into the thigh at the level of the popliteal space and the tibial branch of the sciatic nerve exposed. A No. 26 hypodermic needle was inserted into the nerve for about 1 cm. and 1 to 5 ml. of either iodobenzene or iodophenylundecylate injected into the nerve. Our experience has been that iodobenzene is the better of these two compounds, probably due to its lower viscosity, which enables it to flow more readily. On injection of this substance, it usually follows a single channel in the nerve and only a small part of the nerve is outlined. This is well shown in Fig. 1. Here the injected portion of the nerve stands out as an opaque line. Close examination of this photograph shows that the material has followed the nerve back to the spinal cord and is present in or around the cord.

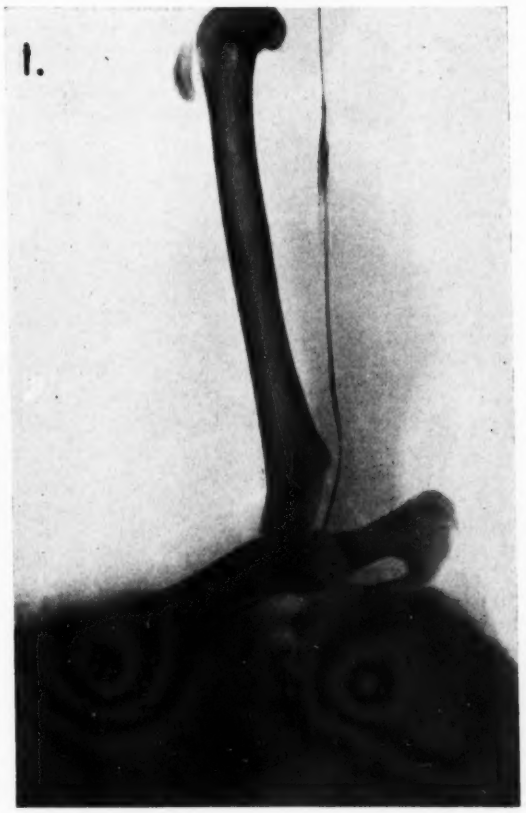

FIG. 1. Roentgenogram of thigh of cat showing the sciatic nerve outlined by injection of iodobenzene.

The technic for making microsecond roentgenograms has been briefly described by Butler *et al.* (1945)[3] and will not be repeated here. However, in all of these earlier experiments, roentgenograms were made with the X-ray beam directed at right angles to the path of the

missile, so that the temporary cavity was always photographed along its long axis. In order to demonstrate the displacement of a nerve by the temporary cavity, it was necessary to mount the gun so as to shoot parallel to the X-ray beam and thus to photograph the temporary cavity across its long axis. The exact manner in which this was accomplished is shown by the diagram in Fig. 2.

With the gun mounted in this position, it is obvious that the missile, after traversing the tissues of the thigh, passed directly into the cassette holding the X-ray film. A detailed description of an inexpensive home-constructed cassette will not be given but it should be added that the cassette and intensifying screens suffered little from the passage of these small missiles and could be used repeatedly.

Conventional roentgenograms of longer exposure were made with a small clinical X-ray outfit, operating at 5 milliamperes, with an average exposure of 4 seconds.

Electrical stimulation of nerves was made with 60 cycle current. The secondary of a "Variac" supplied a low controlled voltage to the primary of a small step-down transformer, which was a radio component filament transformer, and which, in addition to its function as a voltage reducer, served also to isolate from ground the stimulating current that was applied to the nerve. Harvard platinum stimulating hand electrodes were used to apply the

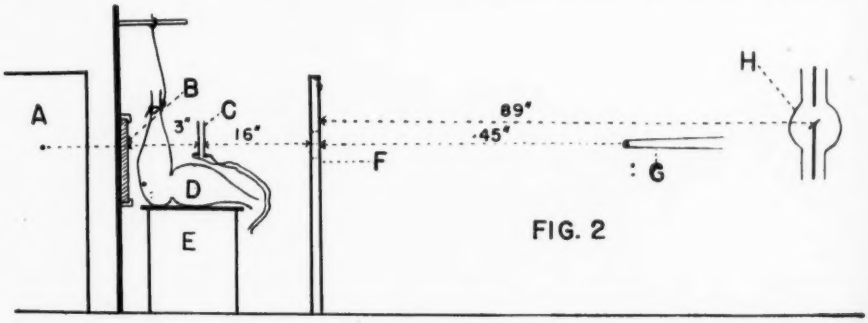

FIG. 2

FIG. 2. Diagram of apparatus used in making microsecond roentgenograms. (A) backstop, (B) cassette holding X-ray film, (C) trigger screen, (D) animal, (E) table supporting animal, (F) sabot screen, (G) gun and (H) X-ray tube.

stimulus to the nerve. The maximal stimulus was of the order of 1 to 2 volts, as tested on undamaged nerves, and the electrical examinations were always concluded with the use of maximal stimuli.

Nerves selected for histological study were fixed in sublimate-formol-acetic mixture. Prior to fixation these segments of the nerve were carefully extended on a flat sheet of cork or on a glass slide. The fixed nerves were sectioned longitudinally at 15 micra. Sections were stained with Delafield's haematoxylin and counter-stained with an eosin-orange "G" mixture.

EXPERIMENTAL RESULTS

1. *Displacement of the Sciatic Nerve by the Temporary Cavity Formed by the Passage of a 4/32" Steel Sphere.* The experiments to be described here are typical of a number that demonstrate clearly the manner in which the sciatic nerve can be displaced by the temporary cavity produced by the passage of a high velocity missile.

Fig. 3 (X-ray #232) is a conventional roentgenogram of the left thigh of a cat before shooting. The sciatic nerve has been made radiopaque by the

injection of several ml. of iodobenzene. The level of injection is indicated by the diffuse dark region where some of the material escaped into the surrounding tissues.

Fig. 4 (X-ray #232) shows a microsecond roentgenogram of this same thigh taken approximately 200 μsec. after the thigh was struck by a 4/32″ steel sphere whose impact velocity was approximately 3,200 ft/sec. This

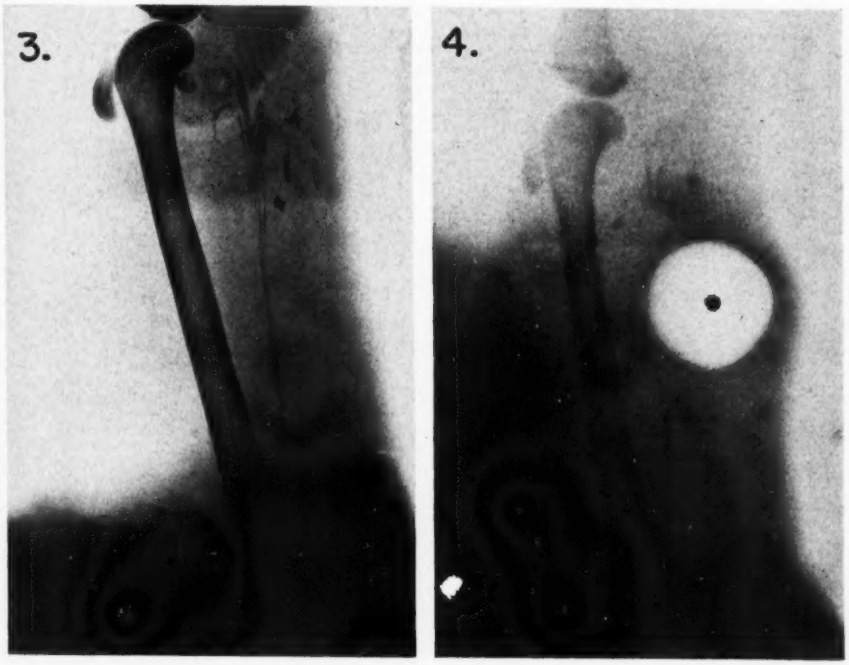

FIG. 3 (*left*). Roentgenogram of the left thigh of a cat showing the sciatic nerve outlined by injection of iodobenzene.

FIG. 4 (*right*). Microsecond roentgenogram of the thigh shown in Fig. 3 taken 200 μsec. after the thigh was struck by a 4/32″ steel sphere whose impact velocity was approximately 3,200 ft/sec. Note the large temporary cavity, circular in cross-section, and the sciatic nerve displaced around its margin.

roentgenogram shows that a temporary cavity is developing in the thigh which, when viewed across its long axis, is approximately circular in configuration.

A close examination of this photograph of the temporary cavity shows a dark area in the center of the cavity. This represents the hole made in the X-ray film by the passage of the missile. The remainder of the cavity area shows a radiation density approximately equal to that of regions outside the thigh and indicates a complete transmission of the radiation through the temporary cavity.

Examination of this roentgenogram also shows that the radiopaque sciatic nerve has been pushed aside as the cavity expands. It is clear that the nerve follows closely the margin of the cavity. Earlier studies lead us to believe that the temporary cavity photographed here has not reached its maximum size and that the nerve will be displaced much more than is evident in this photograph.

Fig. 5 shows a conventional roentgenogram of this same leg, taken im-

FIG. 5 (*left*). Roentgenogram of the thigh shown in Figs. 3 and 4, made immediately after shooting. Note the small permanent cavity and compare with the temporary cavity shown in Fig. 4.

FIG. 6 (*right*). Roentgenogram of the left thigh of a cat showing the sciatic nerve outlined by injection of iodophenylundecylate.

mediately after shooting. In this photograph the residual cavity appears as a small light area approximately 1 cm. from the nerve. The nerve has returned to its normal position as seen in Fig. 3.

A second and similar case is illustrated by the roentgenograms shown in Figs. 6, 7 and 8.

Fig. 6 (X-ray #229) is a roentgenogram of the left thigh of a cat before shooting. The sciatic nerve has been rather irregularly outlined by injecting several ml. of iodophenylundecylate.

Fig. 7 (X-ray #229) shows the temporary cavity formed in this same thigh immediately after the passage of a 4/32″ steel sphere whose impact velocity was approximately 3,200 ft/sec. This cavity was photographed approximately 180 μsec. after the sphere struck the leg and does not represent the maximum size that this cavity will ultimately reach. This photograph again shows that the broad sciatic nerve is being displaced around the mar-

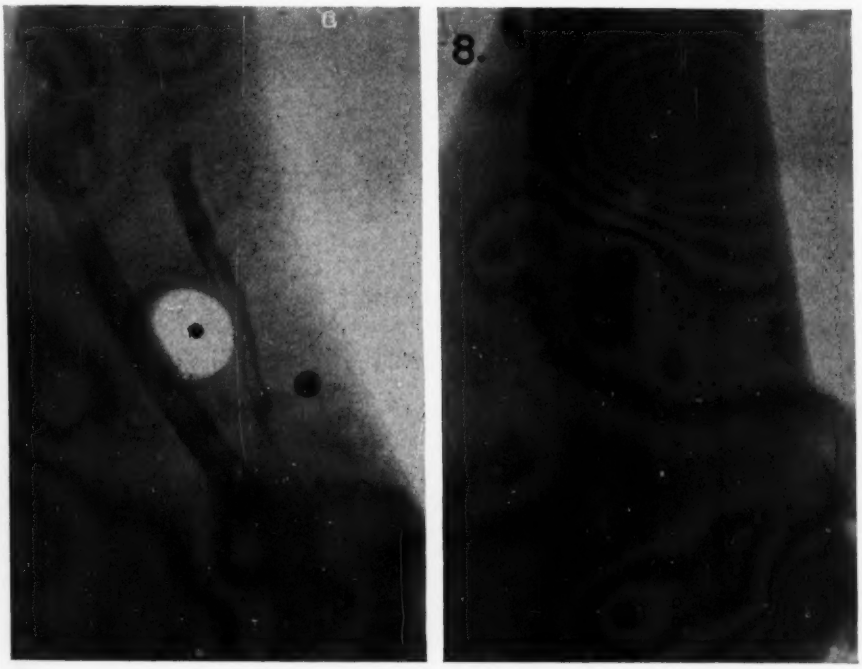

Fig. 7 (*left*). Microsecond roentgenogram of the thigh shown in Fig. 6, taken approximately 180 μsec. after the thigh was struck by a 4/32″ steel sphere whose impact velocity was 3,200 ft/sec. Although the temporary cavity has not reached its maximum size, note the displacement of the sciatic nerve around the margin of the cavity.

Fig. 8 (*right*). Roentgenogram of the thigh shown in Figs. 6 and 7, made immediately after shooting. Note that the femur has been fractured although it was not struck directly by the missile.

gin of the temporary cavity. A small black spot is to be seen just to the right of the nerve in this photograph. This represents the hole made in the intensifying screen by an earlier shot through another thigh.

Fig. 8 (X-ray #229) shows this same thigh, photographed immediately after the shot. It will be noted that the sphere passed almost equidistant between the sciatic nerve and the femur. Although the sphere did not strike the femur directly, it was subjected to such a blow during the development of the temporary cavity that a clean fracture resulted. Examination of the

sciatic nerve reveals that the injection mass has been badly displaced, showing that the nerve has been compressed or stretched. An idea of the force to which the nerve has been subjected can be deduced from the damage to the femur, which lies about equidistant from the path of the missile. It should be borne in mind, however, that the femur is a very rigid structure, while the nerve is quite elastic and free to move.

These two experiments show clearly that the sciatic nerve is moved aside coincident with the expansion of the temporary cavity. It should be emphasized here that this displacement is taking place very rapidly (at a rate approximately 1/10 the velocity of the sphere) and that the nerve is being subjected to stretching and to compression between surrounding tissues. Nerves that have been subjected to this rapid displacement ordinarily show no macroscopic signs of damage, unless directly struck by the missile. Functional damage resulting from such displacement will be described in the next section.

2. *Functional Damage to Nerves—Conduction Studies.* In this series of experiments, missiles were fired into a total of 22 cat thighs. In every case the gun was aimed so that the missile would hit the thigh midway between the knee and hip joint, as close as possible to the sciatic nerve, without actually hitting it. In all cases, microsecond roentgenograms were taken to show the location and size of the temporary cavity, but the nerves were not made radiopaque as very little was known as to the effects of the contrast media on conduction processes.

Immediately after each shot, the sciatic nerve was exposed, initially at the level of the sciatic notch, and then in the course of examination, as far down as the popliteal space. First, examination was made of the functional capacity of the nerve by means of electrical stimulation to determine conduction along the nerve towards the periphery. Whenever functional disturbance was observed, using as indices the responses of various muscles to electrical stimulation of the nerve, the level distal to the point at which the nerve appeared functionally undamaged was carefully explored and the relation of this point to the site of the wound was diagrammed. Final electrical examinations were always made with the nerve cut away from the spinal cord at the level of the sciatic notch, with the muscularis nerve severed and with the nerve raised somewhat away from the tissues of the thigh. These precautions were taken to insure that the observations were not affected by current spread, by reflex activity due to afferent impulses, or by confusion due to the activity of the muscularis nerve and its associated branches, since the muscularis nerve was never affected in any of our experiments.

One of the best indices of conduction in the nerve was the visual observation of activity in the muscles of the toes. The gastrocnemius muscle was also watched. In several cases, visual observation was supplemented with 16 mm. motion picture records.

In several cases where functional damage was evidenced, the animals were kept for 6 hours under nembutal anaesthesia for possible return of

functional activity, but aside from this no prolonged observations were made.

The results of electrical testing of the functional ability of the nerves in the 22 thighs used in these experiments may be summarized as follows:

(1) In *15 cases*, there appeared to be no interference with the ability of the nerve to conduct impulses to the muscles of the lower leg. In practically all of these cases, the missile had passed through the tissues at a distance of more than 2 cm. from the nerve.

(2) In *1 case*, the missile hit the nerve directly and caused complete severance.

(3) In *2 cases*, the missile had severed a portion of the nerve (the tibial bundle) but conduction was absent in the apparently undamaged peroneal bundle as well.

(4) In *4 cases*, the nerve was apparently not hit by the missile, yet conduction was blocked either at the level of the wound track, or slightly above it. Electrical stimulation of the nerve at any point above the block produced no muscle responses, while stimulation immediately below this point gave a normal response.

In all four cases of conduction block the missile had passed within approximately 1 cm. of the nerve. In the 15 cases where conduction was not affected, the missile had usually passed about 1.5 to 2.5 cm. from the nerve. Experiments have shown that the temporary cavity produced by missiles having an impact velocity of 3,000 to 4,000 ft/sec. has a radius of about 2 cm. In three of these latter cases, the missile had passed about 1 cm. from the nerve and in the fourth, about 0.5 cm. In this last case, however, the wound had been in the popliteal space, where the nerve is in such a position that it can undergo considerable displacement. In general, therefore, there seems to be a relationship between the proximity of the missile and the likelihood of its damaging the conductive ability of the nerve. The nerve must lie within the effective radius of the temporary cavity.

A further relation worth noting is that which exists between the level of the wound produced by the missile and the level on the nerve at which a block appeared to have occurred. In three of the four cases where a "clean miss" had caused nerve block, the point of blocking was at the level of the permanent cavity produced by the missile. In the fourth case, the block occurred about 1 cm. proximal to the edge of the permanent cavity. Likewise in the two cases of partial severance of the nerve, the block in the uncut portion of the nerve occurred at the level of the wound in one case and slightly proximal to the wound level in the second. In no case was the block distal to the level of the wound. These observations may be correlated with the position of the nerve in the intact thigh. At the level of the popliteal space, the sciatic nerve lies in a mass of loose connective tissue and takes a curved path, so that on freeing it from the surrounding connective tissues, it may be stretched out considerably in a straight line. Above the popliteal space, the nerve comes to lie in a dense mass of muscle and, when it reaches the level of the sciatic notch, it is relatively strongly fixed in a position, par-

ticularly in view of the bend that the nerve takes over the notch. The nerve must be more subject to damage, therefore, as a result of rapid deformation, stretch or compression in its proximal portion than it is in its distal section.

3. *Histological Studies on Nerves in Which a Conduction Block Had Occurred.* Histological studies were made on the four nerves which were not struck directly by the missiles but which had been found to exhibit functional damage. All four nerves showed no gross signs of damage but microscopic examination revealed structural changes which we feel are the result of wounding. These structural changes were as follows: (1) Large breaks within the nerve, in which the continuity of a large bundle of fibers was broken; (2) a separation of the individual fibers; (3) an extreme "kinking" of the fibers, as though they had undergone abnormal stretching and (4) a series of multiple microscopic injuries scattered along the nerve trunk for a considerable distance. The last-mentioned injuries were found by a critical study of the sections under the oil immersion objective, where it was discovered that the axis cylinders of many fibers were broken but the myelin sheath and neurilemma showed no signs of damage. In all of the sections examined, the nerve sheath (epineurium) appeared undamaged. The accompanying series of photomicrographs shows the nature of this nerve damage.

Fig. 9 shows a longitudinal section of an undamaged control nerve.

Fig. 10 shows a longitudinal section of a nerve (#279) in which conduction was blocked by a near miss and illustrates well certain types of nerve damage. Here it can be seen that the nerve fibers have been widely separated and that many fibers are completely severed, with their ends badly frayed. In other portions of the nerve (Fig. 11), the nerve fibers have been thrown into large kinks, as though they had undergone abnormal stretching.

The criticism may be offered that these areas of damage are not real but merely represent artifacts due to handling and sectioning. A total of six normal control nerves were removed and sectioned in the same manner as the experimental nerves. While certain minor structural changes must inevitably result from handling, none were of the severity described above and we feel that a good part of the damage revealed by our sections must be a direct result of the shot.

DISCUSSION

The experiments just described indicate that a small high velocity missile can cause functional and anatomical damage to a peripheral nerve without making a direct hit upon the nerve and without leaving gross signs of dam-

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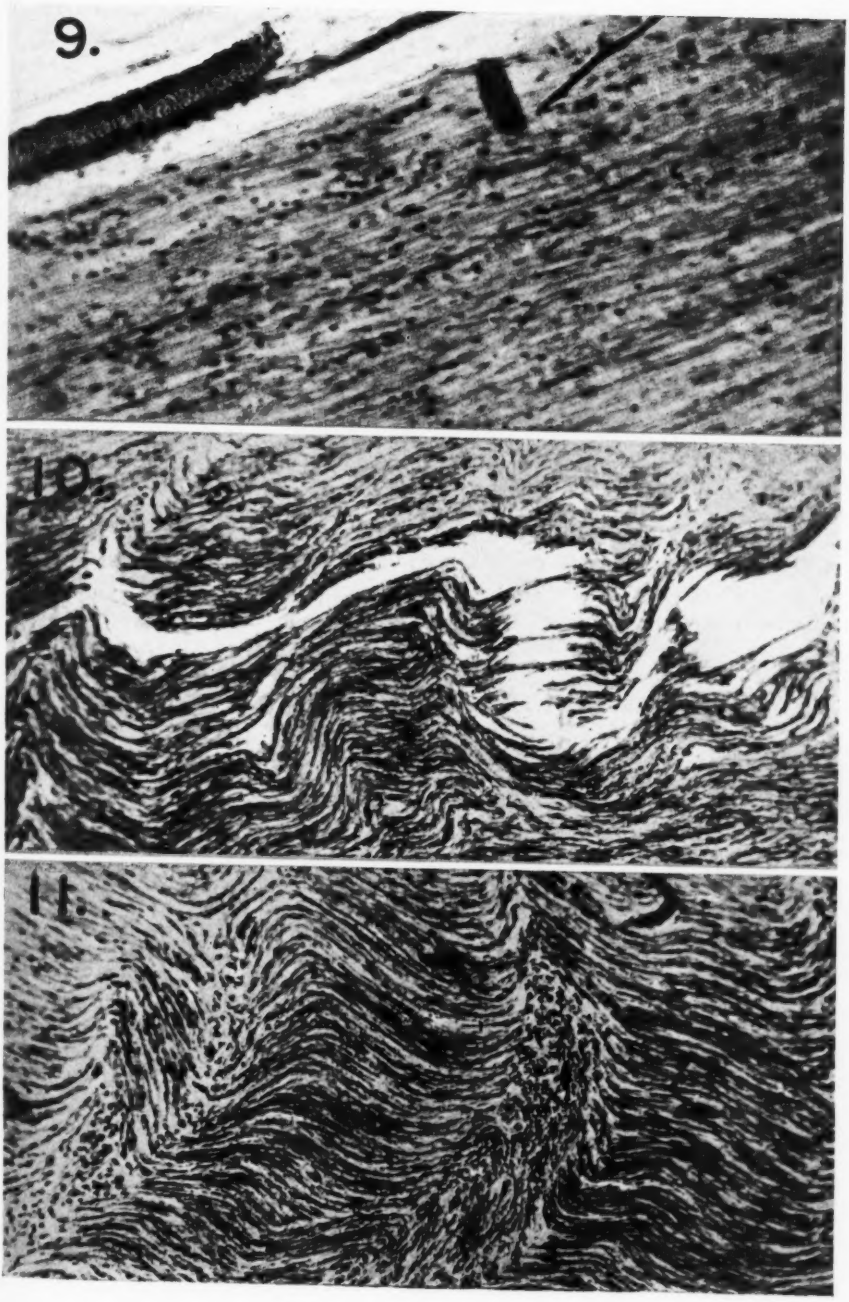
FIG. 9. Photomicrograph (×140) of a longitudinal section of an undamaged control nerve.

FIG. 10. Photomicrograph (×140) of a longitudinal section of a sciatic nerve in which conduction was blocked as a result of a "near miss" by a high velocity 4/32″ steel sphere. Compare with Fig. 9 and note damage to fibers.

FIG. 11. Photomicrograph (×140) of a longitudinal section of a sciatic nerve in which conduction was blocked as a result of a "near miss" by a high velocity 4/32″ steel sphere. Note the manner in which the fibers are thrown in kinks.


9.
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11.

age to the nerve. This structural damage results from the nerve being rapidly tossed aside as the large temporary cavity forms immediately after the passage of a high velocity missile. That such displacement of a nerve actually occurs can be demonstrated by microsecond roentgenograms of cat thighs in which the sciatic nerves have been visualized by the injection of radiopaque media. This rapid displacement can bring about a compression of the nerve between adjacent soft tissues as well as a considerable stretching of the nerve. Such behavior results in a break in continuity of many of the individual nerve fibers. The type of nerve damage found here appears to be very much like that described by Denny-Brown and Brenner (1944)[5] in their experiments dealing with damage to nerves induced by pressure and other agents.

This type of nerve injury may be expected to occur in battle casualties where wounds from high velocity projectiles or bomb fragments are common. Indeed, a series of nerve injuries recently described by Seddon (1943)[11] and Livingston *et al.* (1945)[9] appear to be of this type, where functional damage has been produced without the continuity of the nerve being disrupted and which Seddon calls "axonotmesis."

The importance of recognizing this type of nerve injury cannot be overemphasized. It is well known that nerve injury, such as occurs in crushing a nerve, which does not break the continuity of the nerve sheath, results in a better prognosis than do injuries in which the nerve sheath has been severed and requires suturing (Gutmann, 1942;[7] Neumann *et al.*, 1943;[10] Berry *et al.*, 1944[1]). The difference in behavior of crushed, as compared with severed nerves, is due to the fact that the normal anatomical pathways are more readily established when the nerve sheaths are intact and the approximation of the distal and proximal ends of the severed nerve fibers is more likely than is the case in a completly severed nerve which requires suture.

This paper presents evidence that a good proportion of injuries to peripheral nerves may be produced without severance of, or gross damage to the nerve. It is recommended, therefore, that careful examination of nerve injuries be made prior to drastic operative intervention, bearing in mind the possibility of a functional injury which may result from a series of minimal breaks in the continuity of a nerve. Livingston *et al.* (1945)[9] have come to essentially the same conclusion, as they state: "Unless there is more evidence than a simple loss of function to suggest that a nerve has been severed, a period of observation of 2 to 3 months seems to be indicated in the treatment of nerve lesions caused by high velocity projectile wounding."

SUMMARY

1. The sciatic nerves of living, anaesthetized cats have been visualized by injection of radiopaque media. Microsecond roentgenograms show that the nerve is rapidly blown aside during the expansion of the large temporary cavity which is formed by a high velocity missile which passes near but does not strike the nerve directly.

2. Conduction studies show that functional damage can be produced in nerves that have been subjected to such displacement, even though no break in the continuity of the nerve was produced.

3. Histological studies on sections of these nerves show that the loss of function can be attributed to a series of minor breaks within the nerve sheath.

4. This type of structural damage can be explained by the compression and stretching which the nerve undergoes as it is rapidly blown aside during the expansion of the large temporary cavity which forms in the tissues immediately after the passage of the missile.

5. The importance of recognizing this type of nerve injury before attempting drastic operative measures is emphasized.

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COMPOUND INJURIES OF THE SPINAL CORD*

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THE PROGNOSIS for the return of function following injuries to the spinal cord has been exceedingly poor. Following World War I it was advocated that compound injuries of the spinal cord resulting in complete immediate loss of sensation and motor power below the segment involved be treated conservatively. If the cord had been directly injured by the missile, it was held that surgery would be of no benefit, and merely debridement of the external wound was advised.

The Medical Research Council of Great Britain,[3] in a review of World War I spinal cord injuries, found it simpler to state the contraindications for laminectomy. The only indication for laminectomy was an incomplete lesion with progressive increasing signs of functional loss. They advocated early, but not immediate surgery, stressing a wait of 2 to 6 weeks after the injury or until the spinal shock had ended. The Council did not feel that the initial injury was increased by the presence of a foreign body or indriven bone in the cord. One of the primary contraindications put forth against surgery was the presence of a physiologically complete transverse lesion of 48 hours' duration. In this group of cases the prognosis was said to be hopeless and no interference indicated. Yet it was also stated that in the so-called state of spinal shock there may be no evidence of recovery for days or weeks.

This was the confusion existing when we began to care for compound spinal injuries in World War II. We found the x-ray to be very misleading, and anatomically the degree of damage was far more extensive than the x-ray would indicate. Only gross deformities and severe fractures could be seen on x-ray examination. Indriven bone fragments could not be visualized. Since the most important single factor that came out of this war in the surgical treatment of war wounds and prevention of infection was wound excision and not circumcision, we decided to apply the same principles in compound spinal injuries as those used in other wounds. The only possible way to do an adequate debridement is by a formal laminectomy. Moreover, the presence of foreign bodies or indriven bone in the spinal cord does set up a series of pathological changes. These changes are similar to those produced by the presence of foreign material in the brain. Hassin[1] reported the cellular reaction following the experimental insertion of cotton in the dog brain. He found lymphocytes, plasma cells, fibroblasts and giant cells. The

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foreign body is surrounded by fibroblasts and these large giant cells. Lichtenstein and Kirshbaum[2] found a similar reaction to a foreign body in the spinal cord. They report the changes found in an infant's spinal cord, the foreign material being hair. Here also fibroblasts and giant cells were the predominant features with the addition of dense masses of collagenous connective tissue.

It is felt that the removal of irritant foreign material prevents the development of the above reactions. In addition, the laminectomy allows for repair of the dural defect and the reestablishment of normal circulation in and about the cord. Thus in this seemingly hopeless series of cases an attempt would be made to allow for the maximum return of function of any remaining viable spinal cord structures.

TABLE 1

Compound spinal injuries in this series

	Immediate Incomplete Paralysis	Immediate Complete Paralysis	Total
No. of cases	3	19	22
Infections	0	0	0
Improved	3	2	5
Deaths	0	1	1

Twenty-two compound spinal injuries were operated upon in this series. Table 1 shows that there was only one death.

The death was that of a 25-year-old Mexican soldier who had a large shell fragment in the spinal canal at the level of C-6. It measured 2×1 inches and was corkscrew in shape. When seen the patient's condition was very poor, his blood pressure was unobtainable, and he was cold and clammy, but conscious. He had evidence of a complete lesion at the level of C-6. He was given 3 units of plasma and 1,500 cc. of whole blood. The blood pressure came up to 78 systolic, but no diastolic could be obtained. He remained in this condition for approximately 4 hours, and, since there was no improvement, it was decided to do a laminectomy. The operation revealed that the entire cord had been severed by this large foreign body in the canal. He remained cold and clammy with a blood pressure which rarely went over 70, and he expired on the 3rd postoperative day.

Table 1 shows further that there were no postoperative infections. The patients who had an incomplete lesion on admission did very well postoperatively, with function returning rapidly. Of those with a complete lesion, two showed remarkable return of function, and are being reported here.

Case 1. This soldier, age 23, had been wounded on September 27 at 1115 hr. by fragments from a high explosive shell. The force of the explosion threw him to the ground; however, there was no loss of consciousness. He was immediately aware of complete numbness from the waist down, inability to move his legs, difficulty in moving his arms, and numbness along the ulnar distributions of both arms. He was seen at 0800 hr., September 28, 21 hrs. after the injury. He was conscious and there was no evidence of shock. At that time there was evidence of a complete transverse lesion at C-7, loss of sensation in all modalities, and a flaccid paraplegia. There was weakness in hand grasp bilaterally and in extension of both forearms. The biceps reflex could be obtained, but was diminished. No reflexes were obtain-

able in the lower extremities and the plantar responses were neutral. He had complete urinary retention. The wound of entrance was small, measuring 4 × 4 cm. It was to the right of the midline at the level of C-6. X-ray revealed many small foreign bodies to the left of the midline from C-7 to T-1. There was a small foreign body in the region of the cord at C-7. The laminae of C-6, C-7, and T-1 were comminuted on the right.

His general condition was good and he was prepared for laminectomy at 0900 hr., September 28. The operation was done under local procaine. The wound of entrance was excised down to the laminae. A midline incision was used to expose the involved vertebra. The spines and laminae of C-6, C-7 and T-1 were removed. At the level of C-7 there was marked comminution of the lamina with indriven bone lacerating the dura. On the left many small foreign bodies were found and removed. The dura was opened widely and the cord inspected. The cord was somewhat pale and at C-7 a small pin-point penetration of the cord was seen. No attempt was made to remove this foreign body. The dura was repaired and closed tightly. The midline incision was closed in layers and the wound of entrance left open. Suprapubic drainage was instituted.

Postoperatively he did very well. While under my observation he showed no return of function. He was evacuated to a general hospital on the 6th postoperative day. The midline incision had healed by first intention. The wound of entrance was granulating nicely. He was not seen again until October 12, or 16 days after injury. Three days after arriving at the general hospital he began to show return of function. On October 12, when seen by me, he was moving his toes and feet, position sense and touch were returning, there was a patchy return of sensation to pin prick, reflexes were hyperactive in the lower extremities, and the plantar responses were still neutral. He was still on suprapubic drainage.

Case 2. This soldier, age 27 years, was injured on September 27, at 1500 hr. and was seen on September 28 at 1635 hr., or 25 hrs. after injury. After being struck by a missile from a high explosive shell he was thrown to the ground, but did not lose consciousness. There was immediate numbness of both lower extremities with inability to move them. On examination he was in good physical condition and he had a complete level at T-11. X-rays revealed a missile in the canal at the level of T-12, measuring 5 × 2 cm. The wound of entrance was on the right side, 1½ inches from the midline.

He was operated upon on September 28 at 1800 hr. The wound of entrance was excised down to the lamina, and a midline incision was made to expose T-11, T-12 and L-1 vertebrae. The laminae on the right side in the region of T-12 and L-1 were shattered by the missile which was lodged in the canal. The spines and laminae of T-11, T-12 and L-1 were removed. The dura on exposure was found to be destroyed from the level of T-11 to L-1. On removing the missile from the canal, a cavity in the cord, measuring 5 × 5 mm., was left. There was much dirt and clothing that had been indriven with the missile and was lodged in the muscles on the right side. All this debris was removed. The cord, other than the small cavity left by the missile, did not appear to be damaged. A fascial graft was used for repair of the dura. After closing the dura, the midline incision was closed in layers and the lateral wound was left opened. Suprapubic drainage was instituted.

Three days postoperatively the patient began to show signs of returning function. There was a return of position sense and he began to wiggle his toes. He was seen on October 20, 23 days after his injury, at a general hospital. He was urinating spontaneously and moving his legs up to his knees. Sensation had returned to pin prick and touch, although it was patchy and incomplete.

Whether or not these two patients would have recovered without laminectomy is a moot question. The removal of indriven bone, foreign bodies or debris from the region of the spinal cord at the earliest possible time may have influenced the recovery. A new aspect is presented here—the presence

of an immediate complete transverse lesion does not necessitate a hopeless outcome.

CONCLUSIONS

Compound spinal wounds should be treated like any other wound. It is essential to do a complete toilet of the wound and only a laminectomy will accomplish this.

Personal communications from men who have followed these cases in general hospitals here in the United States, indicate that more intelligent management can be undertaken when the pathology has been observed and recorded. It is further stated by these men that the patient does better from a nutritional and physiological standpoint if a complete debridement, or in other words a laminectomy, has been performed early.

A laminectomy performed by a competent surgeon does not increase the mortality and cuts down the dangers of infection.

The above two cases are a ray of light in a depressing group of patients. The presence of a complete immediate paralysis does not indicate a hopeless situation. It is felt that if the patient's condition is good, early surgery will give the patient the best possible chance for functional recovery of any remaining viable spinal cord.

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EXPERIMENTAL OBSERVATIONS ON THE USE OF STAINLESS STEEL FOR CRANIOPLASTY

A COMPARISON WITH TANTALUM*

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DURING the past war many articles[4,5,6,7,8,10,11] were published on the use of tantalum for cranioplasty. To date the results have been satisfactory. However, the writers believed that it might be worth while to investigate the use of stainless steel in animal and human cranioplasties since the material has been used successfully for many years in various parts of the body. It is easily shaped and cut at the operating table, and its present cost is 1/290th of tantalum.

The use of stainless steel as suture material was first introduced by Babcock (1935), who showed that it evokes minimal tissue reaction as compared with catgut sutures.[1]

The authors, as well as others, have found stainless steel wire very satisfactory for closure of laminectomies. Orthopedic surgeons have used it for bone plates and screws.

We can find no reference in literature concerning histological studies following the use of stainless steel plates in cranioplasties. Boldrey[2] has used stainless steel wire mesh for the closure of small cranial defects. He placed a layer over the external table of the skull. No evidence of irritation of the meninges or brain was found, and fibrous and bony connective tissue practically welded the two wire-mesh layers into one firm plate. Chao, Humphreys and Penfield (1940)[3] investigated the reaction following the use of stainless steel plates (as well as other metallic substances), in the subdural space for the prevention of meningocerebral adhesions. These authors make no mention of its use for cranioplasties.

The present communication deals with repair of cranial defects in dogs, using perforated, stainless steel plates.†

The studies were carried out on three dogs, which were sacrificed at the end of six weeks, three months, and six months respectively.

Dog #1. Under intravenous nembutal a right parietofrontal craniotomy was performed. The dura was left open and the bone defect was repaired by an overlying stainless steel plate, $3 \times \frac{1}{2}$ cm. The temporal muscle was sutured over the plate with stainless steel wire sutures, and the skin closed. Wound healing was normal. After 6 weeks the animal was sacrificed. There was no fluid above or below the plate. After reflection of the temporal muscle

* Presented before the Philadelphia Neurological Society, October 26, 1945.

† Analysis and thickness: The stainless steel plate was 0.015 inches in thickness. The stainless steel used had the following composition: carbon, .08–.20%; manganese, 1.25% maximum; silicon, .75% maximum; sulfur and phosphorus, .03% maximum; chromium, 18–20%; and nickel, 8–10%.

the plate was found to be completely encapsulated by a thickened, smooth, glistening hyaline membrane.

Microscopic. Sections of this membrane did not reveal the presence of any inflammatory reaction. The membrane resembled a thickened dura. No photomicrographs were available for reproduction.

Dog #2. A bilateral trephine opening was made over both parietal areas. The dura was opened on the right and a small, perforated, stainless steel plate, 1½ cm. square, was placed beneath it in the subdural space and the dura closed with stainless steel wire. The muscle was closed with stainless steel wire. The dura was not opened on the left. A perforated stainless steel plate was anchored to the bone over the trephine opening by stainless steel wire. Healing was excellent and at no time were any collections of fluid noticed beneath the muscle or scalp. Three months later the animal was sacrificed by intravenous nembutal.

FIG. 1 (*left*). Dog #2. The temporal muscle is reflected, showing the stainless steel plate surrounded by a thin, transparent capsule.

FIG. 2 (*right*). Dog #2. The outer capsular layer is reflected, exposing the underlying, shiny, stainless steel plate. No fluid was found above or below the plate.

Autopsy. Left trephine (plate sutured to external table around trephine opening). There was no serous collection beneath the scalp or temporal muscle. The undersurface of the muscle was adherent to a very thin, transparent capsule which covered the outer surface of the plate (Fig. 1). The capsule, approximately 1 mm. in thickness, was incised and reflected from the underlying shiny plate. The undersurface of this membrane was glistening (Fig. 2) and no fluid was found between it and the plate. The plate was completely encapsulated, and bands of firm tissue penetrating the perforations in it firmly united the outer and inner layer of the capsule. A section of this capsule, and overlying muscle, showed no inflammatory reaction (Fig. 3A). The plate was firmly anchored to the bone and upon elevation its undersurface was just as clean and shiny as the outer surface. There was no fluid beneath the plate. The bony defect was completely covered by glistening white tissue, the outermost layer of which was apparently the inner layer of capsule surrounding the plate. Sections of this tissue showed the absence of inflammatory reaction (Fig. 3B). The inner layer of the capsule was composed of a thickened layer of fibrous tissue consisting of numerous fibroblasts. The nuclei of the fibroblasts were arranged parallel to the surface of the capsule. The dura was flat, of normal color, and, although thickened, showed no inflammatory reaction (Fig. 3C). The bony edges of the trephine openings were smooth and showed no abnormal reaction.

Right trephine (subdural insertion of plate). The temporal muscle was reflected, exposing the dura. The dura was reflected laterally and a capsule was found adherent to its undersurface. The capsule was approximately 1 mm. thick and its undersurface was smooth and glistening, where it had been applied to the outer surface of the plate. Firm tissue had grown

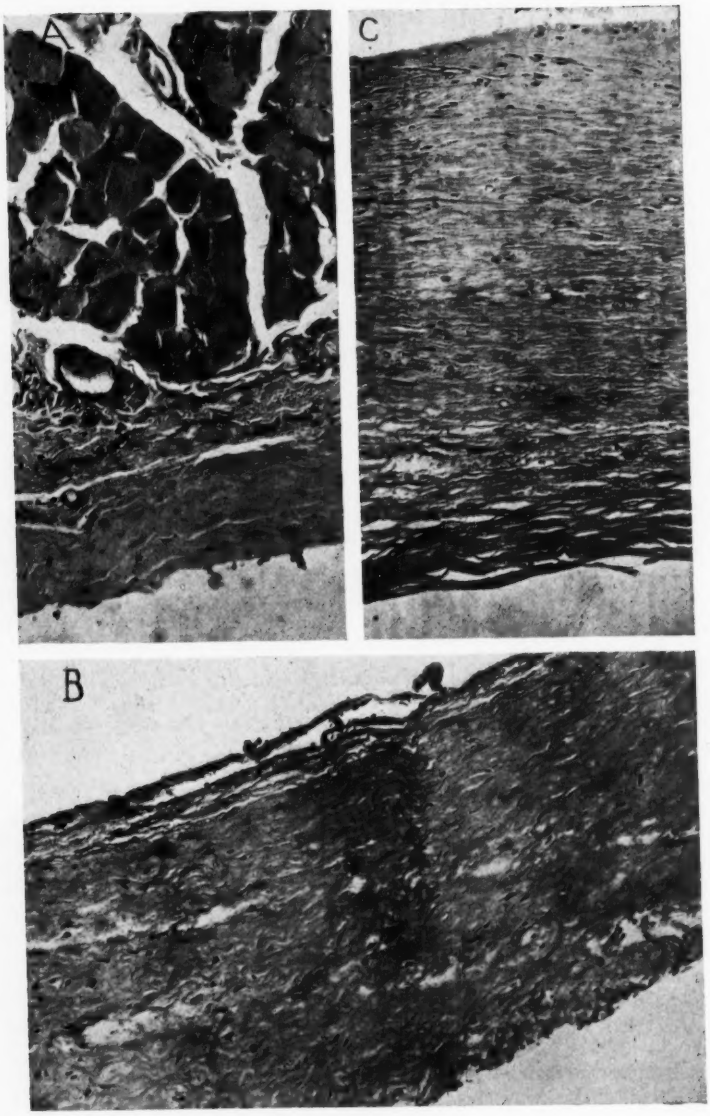

FIG. 3. Dog #2. (A) The temporal muscle is adherent to the outer capsule, which resembles a thickened dura. This outer capsule was in contact with the outer surface of the stainless steel plate. (B) Inner layer of the capsule surrounding the stainless steel plate. (C) Thickened dura beneath the inner capsular layer. Note the absence of inflammatory reaction.

through the perforations in the plate and firmly united the outer and inner layers of the capsule. The plate was removed, revealing the inner layer of the capsule (Fig. 4). It was therefore apparent that the entire plate that was inserted into the subdural space was completely encapsulated. The inner layer of the capsule was thinner than the outer layer. The outer surface of the inner capsule was smooth and glistening and the undersurface of the inner capsule was attached to the underlying arachnoid by very fine, fibrinous adhesions which

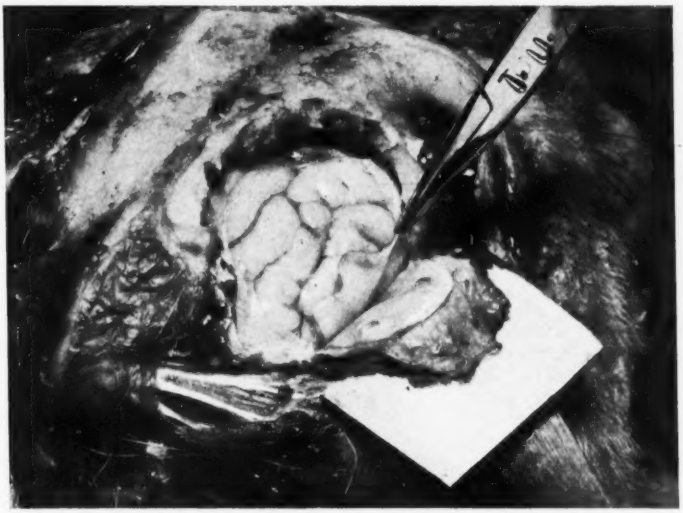

FIG. 4. Dog #2. Right-sided subdural insertion of stainless steel plate. The plate has been removed, revealing inner and outer capsular layers. The inner capsular layer adjacent to the pia arachnoid is grasped with a hemostat.

could be easily broken. Fig. 4 shows this layer separated from the arachnoid. The cortex showed an impression of the overlying plate. A block section was removed for study consisting of dura, both capsule layers, and underlying cerebral cortex (Fig. 5 A, B, C). The outer and inner layers resembled thickened dura. No inflammatory reaction was seen. The brain presented a normal appearance. The pia mater and the pia arachnoid were fused and showed slight increase in cellular content, but no inflammatory reaction was noted.

Dog #3. A small trephine opening was made over the right temporoparietal area and the dura was not opened. A small, perforated stainless steel plate, 2×2 cm., was placed over the bony defect but was not anchored to the bone. The muscle layer was sutured over the plate. Healing was uneventful and the animal was sacrificed 6 months later. The incision healed well and no fluctuation was noted beneath the scalp. The temporal muscle was firmly adherent to the outer layer of the capsule, which, in turn, was firmly adherent by prolongations of tissue through the perforations in the plate to the inner layer of the capsule. There was no free fluid. Although the plate was not wired it was in good position and very firmly fixed by the surrounding tissue. The plate was shiny, and after removal the inner capsule was found to be smooth and glistening. The bony defect was completely healed in with fibrous tissue. Sections of the inner and outer layers of the capsule were studied histologically. Again the layer was found to resemble a thickened dura without evidence of any inflammatory reaction (Fig. 6 A, B).

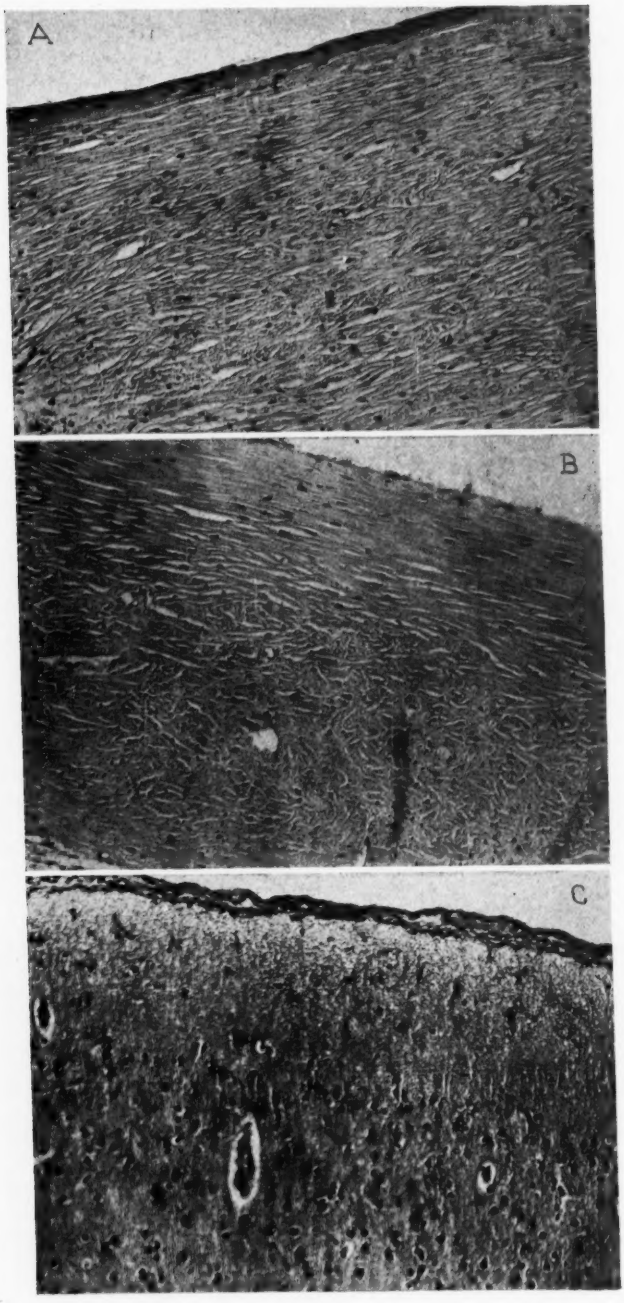

FIG. 5. See opposite page for description.

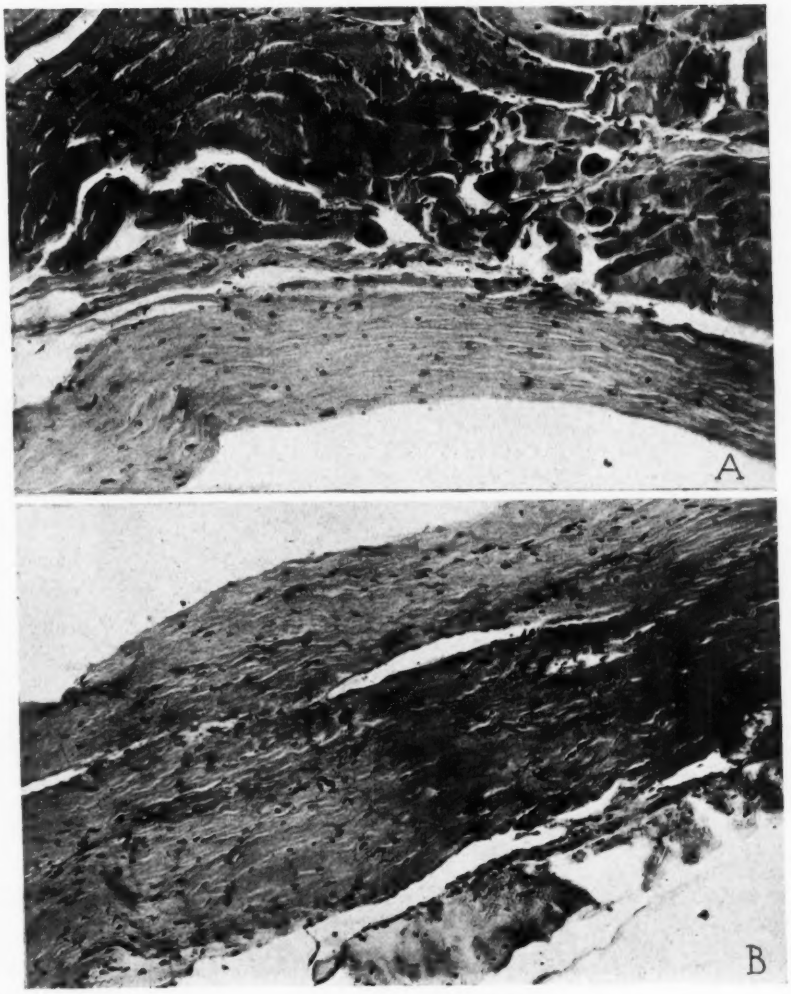

FIG. 6. Dog #3. (A) The temporal muscle is adherent to the glistening outer capsular layer immediately above the stainless steel plate. (B) Inner capsular layer immediately below the stainless steel plate but above the intact dura. The tissue resembles a hypertrophied dura.

FIG. 5. Dog #2. Right-sided subdural insertion of stainless steel plate. (A) Outer capsular layer, which resembles thickened dura. Note the absence of inflammatory reaction. (B) Inner capsular layer adjacent to the pia arachnoid. The tissue resembles hypertrophied dura. (C) Cortex beneath the plate. The cortex approximates a normal appearance. The pia mater and pia arachnoid are fused and thickened.

COMMENT

The gross and microscopic studies made in three dogs showed that the stainless steel plates were completely encapsulated in every instance. There was no evidence of infiltration by polymorphonuclear leucocytes or round cells in any of the sections. There was no collection of serum above or below the plates. The reaction following the insertion of a stainless steel plate in the subdural space compared favorably with that reported by Pudenz and Odom[9] following insertion of a tantalum plate in the subdural space.

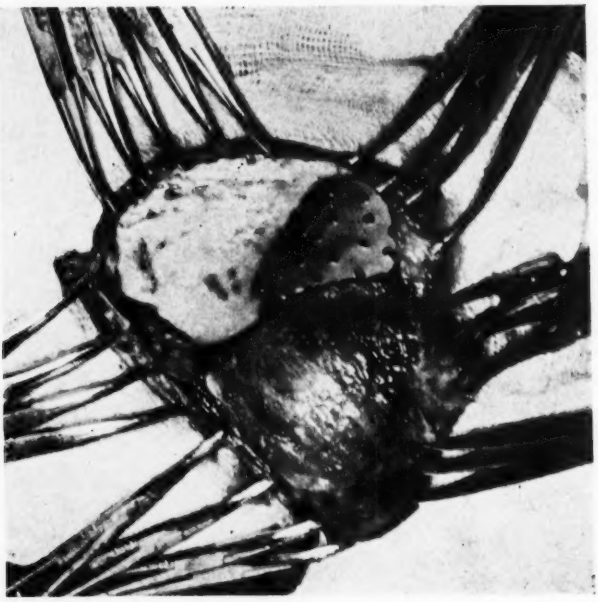

FIG. 7. Repair of a defect in the lateral orbital wall in a human. The temporal muscle is reflected, exposing the perforated stainless steel plate.

We have used stainless steel plates in repair of cranial defects in three patients, but postoperatively the follow-up period is too short to warrant any conclusions. Fig. 7 shows a stainless steel perforated plate* inserted in a defect in the lateral orbital wall following decompression for malignant exophthalmos. The temporal muscle covered the perforated plate.†

* Stainless steel used in repair of human skulls was the same thickness as that used in the experimental work (0.015 inch).

† Dr. Rudolph Jaeger of Philadelphia, in his discussion of this paper, reported a case in which he had placed a huge stainless steel plate over the top of the skull following the removal of a meningioma. The procedure was done approximately 6 years ago. Reexamination of the patient a year ago (5 years later) showed excellent healing.

CONCLUSIONS

The reactions in dogs following insertion of stainless steel plates for cranioplasty and in the subdural space compare favorably with reports in literature concerning reactions to tantalum plates in similar locations. Since stainless steel plates are easily obtainable, can be cut, shaped, and molded at the operating table, and are approximately 1/290th the price of tantalum,* the authors believe that this material should be given a thorough clinical trial.

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* The price of a tantalum plate, 6×6×0.015 inches, is $29.40. The price of a stainless steel plate, 6×6×0.015 inches, is ten cents.

FAILURE IN EARLY SECONDARY REPAIR OF SKULL DEFECTS WITH TANTALUM*

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THE INTRODUCTION of tantalum for the repair of cranial defects has been very gratifying to the neurological surgeon. The highly satisfactory results which have proved possible in the last few years are attributable, in part, to the parallel development of drugs that prevent or arrest infection—the sulfa drugs and penicillin. We know that in the past under ideal circumstances foreign bodies such as boiled bone, rubber or celluloid have been tolerated by tissues without the use of specific drugs. Experience with combat wounds has shown complete (and perhaps permanent) healing of wounds containing large quantities of clothing, hair and metallic fragments. One can hardly question that healing was made possible by the use of penicillin and the sulfa drugs. These facts are mentioned since it is only fair in comparing tantalum with its predecessors, which would doubtless have proved more successful with the addition of our present means of combating infection.

It has been conventional neurosurgical practice to replace separated bone fragments when a depressed skull fracture has been debrided and closed within 8 hours of injury, if operation has converted the wound to a clean one. The "safe period" has been lengthened under favorable circumstances. The use of tantalum rather than bone replacement in the primary repair of cranial defects probably does not increase the risk. However, when primary closure is performed late, neither separated bone fragments nor tantalum should be placed in the wound to jeopardize healing.

The favorable results, then, in primary plating[1] are in accord with proved surgical principles. But this useful addition to our surgical armamentarium must not mislead ardent surgeons to perform secondary plating under unfavorable conditions.

The secondary repair of skull defects is an elective procedure in all respects. Correspondingly, all established principles must be observed. Complete neurosurgical facilities must be available since no intracranial procedure should be undertaken without allowing for the possibility of encountering unexpected obstacles. Tropical climates are undesirable because of the difficulties of wound healing and at times the unfavorable general condition of the patient. Above all, sufficient time must have elapsed after complete

* The opinions expressed in this article are the private views of the authors and are not to be construed as official or reflecting the views of the Navy Department.

† Released from military service. Now at 2915 San Jacinto Street, Houston 4, Texas.

wound healing. Secondary plating must not be compared with the more immediate, necessary measures such as foreign body removal and excision of osteomyelitis which are required for healing of the wound and which lessen the possibility of brain abscess and meningitis in patients with open head wounds.

Adequate time for the healing of a wound before a reconstructive procedure such as plating is performed accomplishes two important things. First, time is given for the many small foci of bacteria, enmeshed, but still viable, to be largely or completely destroyed by the healing tissues. Probably recrudescences of infection are as much or more to be feared when wounds have healed with the help of anti-bacterial drugs. Organisms potentially dangerous, but insensitive to the available drugs, may be among those present and capable of a devastating spread if the natural barriers are prematurely broken. Second, healing tissues increase greatly in strength and mobility when allowed adequate time. Wounds that would have required extensive relaxing incisions while the scalp was friable in the premature stages of healing may be closed with ease after sufficient time has increased the mobility and strength of the layers. The best practices in plastic surgery, employing primary skin grafting of uncovered areas, are utilized when complete closure is otherwise impossible, but must not be resorted to when they would be made unnecessary by a wait of a few months. Any added incision must be made only after careful appraisal of its effect upon the blood supply and upon the subsequent reconstructive procedure.

In the 8 cases reported here, secondary plating with tantalum was attempted in a forward area under unfavorable conditions. Only 2 patients successfully plated under similar circumstances were seen at this hospital during the period in which these 8 patients were evacuated.

REPORT OF CASES THAT REQUIRED REMOVAL OF TANTALUM PLATES

Case 1. This 32-year-old man was wounded by shell fragments on 3 May 1945, at Okinawa, sustaining a compound cranio-cerebral wound of the left occipital area. Initial debridement was carried out the same day. He was transferred on 9 May to an advanced Naval Base Hospital. Neurological examination was negative except for serious bilateral impairment of vision. The optic discs showed papilledema. On 16 May a combined wound debridement and tantalum cranioplasty was carried out. An occipital lobe abscess containing 60 cc. of pus was entered and drained, and a tantalum plate was inserted to prevent additional cerebral herniation. On 15 August a secondary suture was done to obtain closure over the plate.

He was admitted to this hospital on 11 September 1945, with two granulating areas extending from the margins of the scalp defect over the tantalum plate. On 26 September the plate was removed. There was a thick layer of unhealthy granulation tissue beneath the scalp overlying the tantalum plate. Wound culture showed *Staphylococcus albus.*

Case 2. A man aged 29 years was wounded by shell fragments on 9 March 1945, at Iwo Jima, sustaining a compound cranio-cerebral wound of the left temporoparietal area. He was transferred on 12 March to an advanced Naval Base Hospital in critical condition with marked right hemiplegia and aphasia. Debridement of necrotic scalp, bone, and brain was carried out on 21 March. On 9 July a combined debridement and tantalum cranioplasty was

done. It was noted at surgery that the wound contained foul yellowish exudate. The scalp could not be fully closed over the plate. On 21 August and again on 11 September attempts were made to obtain closure by shifting a pedicle graft of scalp and by secondary suture.

He was admitted to this hospital on 23 October 1945, with a 3-inch scalp defect exposing the tantalum plate (Fig. 1). The plate was removed on 3 November. There were thick unhealthy granulations both superficial and deep to the plate. Wound culture showed hemolytic *Staphylococcus aureus*.

FIG. 1. Case 2. This photograph reveals the exposed tantalum plate and the epithelialized area from which the sliding graft had been taken.

Case 3. This 22-year-old male was wounded by shell fragments on 4 May 1945, at Okinawa, sustaining a compound cranio-cerebral wound of the left parieto-occipital area. Initial debridement was carried out on 6 May. He was transferred on 30 May to an advanced Naval Base Hospital with a granulating cerebral wound 3 × 2 cm. Neurological examination showed a left homonymous hemianopsia but no paralysis. By 6 July the wound had healed secondarily. On 9 July a combined debridement and tantalum cranioplasty was performed. The wound was drained. Healing was incomplete, and on 11 September resuture was done in an attempt to obtain closure over the plate.

He was admitted to this hospital on 23 October 1945, with a large scalp defect exposing the tantalum plate. On 1 November the plate was removed. There were thick unhealthy granulations both superficial and deep to the tantalum plate. Wound culture showed hemolytic *Staphylococcus aureus*.

Case 4. A 20-year-old man was wounded by shell fragments on 9 May 1945, at Okinawa, sustaining a compound cranio-cerebral wound of the left frontotemporal area. On 10 May initial debridement was done. The dura mater was completely closed. He was transferred on

22 May to an advanced Naval Base Hospital in good condition and without neurological defect. The wound was granulating, and on 12 July was reported healed. On 18 July combined debridement and tantalum cranioplasty was done. Healing was incomplete. A large pedicle scalp flap was shifted on 20 August in an attempt to close a 3×2 cm. defect overlying the tantalum plate. On 11 September an additional debridement and suture of the wound was done.

He was admitted to this hospital on 23 October 1945, with a 6-cm. scalp defect exposing the tantalum plate (Fig. 2). There was an additional large granulating scalp defect at the vertex from which the pedicle flap had been shifted. On 31 October the tantalum plate was

FIG. 2. Case 4. The photograph reveals the exposed tantalum plate and the epithelialized area where a relaxing incision had been made.

removed. There were thick unhealthy granulations both superficial and deep to the plate. Wound culture showed hemolytic *Staphylococcus aureus*.

Case 5. A man aged 21 years was wounded by shell fragments on 24 June 1945, at Okinawa, sustaining a compound cranio-cerebral wound of the right fronto-temporo-parietal area. Initial debridement was done within 24 hours. He was transferred on 29 June to an advanced Naval Base Hospital with a granulating cerebral wound about 6×3 cm. in area. Neurological examination was negative. On 12 July he was taken to surgery because of convulsions followed by unconsciousness. Two cerebral abscesses were encountered and drained. A tantalum plate was inserted. The wound continued to drain postoperatively, and two attempts were made to close the defect by resuture of the wound.

He was admitted to this hospital on 25 October 1945, with a scalp defect 3×2 cm. in area overlying the tantalum plate. On 1 November the plate was removed. There were thick unhealthy granulations both superficial and deep to the plate, and several collections of fluid pus beneath the plate. Culture showed hemolytic *Staphylococcus aureus*.

Case 6. This 25-year-old man was wounded by a bullet on 18 June 1945, at Okinawa, sustaining a compound cranio-cerebral wound of the right temporo-occipital area. The in-

itial debridement was done within 24 hours. He was transferred on 5 July to an advanced Naval Base Hospital. Examination showed left homonymous hemianopsia and slight left hemiparesis. On 26 July a combined debridement (including drainage of a "walled-off subdural abscess") and tantalum cranioplasty was done. The wound did not heal completely and on 11 September secondary suture was carried out.

He was admitted to this hospital on 25 October 1945. The plate was removed on 2 November. There were thick unhealthy granulations both superficial and deep to the plate. Wound culture showed hemolytic *Staphylococcus aureus*.

Case 7. This 20-year-old male was wounded by shell fragments on 13 May 1945, at Okinawa, sustaining a compound cranio-cerebral wound of the right frontotemporal area. Initial debridement was carried out the same day. On 30 May he was admitted to an advanced Naval Base Hospital. Neurological examination showed a moderate left hemiparesis. On 27 August a combined debridement and tantalum cranioplasty was carried out. Healing was incomplete and on 11 September resuture was done in an attempt to obtain closure over the plate.

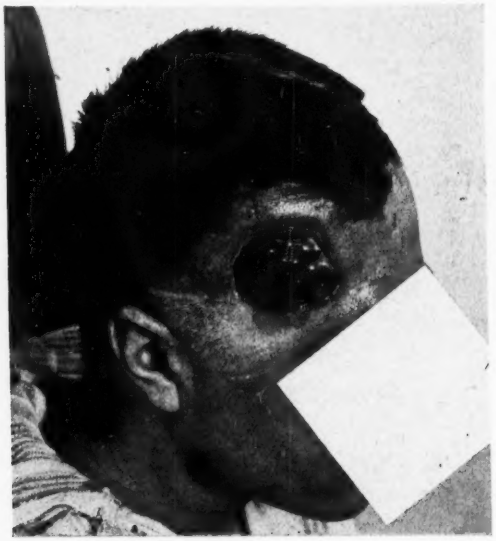

FIG. 3. Case 7. In this photograph the wide expanse of tantalum plate and the epithelialized defect from the relaxing incision can be seen.

He was admitted to this hospital on 30 October 1945, with a scalp defect, 5 cm. in diameter, overlying the tantalum plate (Fig. 3). The plate was removed on 26 November. There were thick granulations both superficial and deep to the plate, and about 60 cc. of fluid pus escaped from beneath the plate as it was lifted out. Both hemolytic *Staphylococcus aureus* and anaerobic *Streptococcus* were cultured from the exudate.

Case 8. A man aged 22 years was wounded by shell fragments on 22 June 1945, at Okinawa, sustaining a compound cranio-cerebral wound of the left fronto-temporo-parietal area. No interval health record is available.

He was admitted to this hospital on 22 November 1945, with extensive traumatic and

surgical scarring of the entire left scalp. The underlying tantalum plate was exposed in three areas (Fig. 4). There was a large granulating defect at the vertex from which a pedicle scalp flap had been shifted. On 27 November the tantalum plate was removed. Beneath the plate there were thick unhealthy granulations, and about 35 cc. of thick pus. Culture showed both *Staphylococcus albus* and *Streptococcus*.

OPERATIVE PROCEDURE

The operative procedure for the removal of the tantalum plates in these 8 patients has been uniform except for the management of the individual problems offered. No new incisions were made. Old incision lines and friable scalp margins were excised. The bone edges were usually healthy in appearance and were shown to be well vascularized by sampling the margins with the rongeur in some instances. The granulation tissue both beneath the galea apo-

FIG. 4. Case 8. This photograph shows, in front of the ear, the exposed tantalum plate, and, above, the incompletely epithelialized defect from which a sliding graft had been taken.

neurotica and upon the dura mater was removed. Contracted scar tissue was excised in order to mobilize the scalp margins and minimize the subsequent scalp defect. An essentially complete closure was made in all instances with a single layer of interrupted sutures, many of which were vertical or horizontal mattress sutures. A No. 14 catheter was placed throughout the length of the wound, with its exit at the point nearest the occiput. This sufficed both for drainage and for irrigation with penicillin solution (1,000 units per cc.). Usually this was left in place for 5 to 10 days and irrigated at 6-hour intervals. This method of closure has minimized both the time of healing and the degree of separation of the wound margins. Probably in most of these patients plating can be satisfactorily performed after a year's interval in a single operation.

STATISTICAL SUMMARY

With two exceptions (Cases 2 and 3) the initial debridement was carried out within 24 hours of wounding. Case 3 was debrided within 48 hours; Case 2 was not debrided until 12 days after wounding. At the time of initial debridement complete closure of the dura mater was obtained in only one patient (Case 4) and even then scalp closure was not complete. The remaining 7 did not have either dural or scalp closure at the time of primary operation.

The interval between wounding and the insertion of the tantalum plate varied from 13 days (Case 1), to 4 months (Case 2), with an average of 56 days. The average interval from insertion of the plate to its removal was 4.3 months. The plates were removed on an average of 10 days after admission to this hospital.

In two patients (Cases 3 and 4) the wound had healed secondarily at the time the tantalum plate was placed. One had been healed for 3 days and the other for 6 days at the time of operation. In the remaining 6 cases tantalum cranioplasty was performed prior to wound healing. In one instance (Case 1) it was stated by the surgeon that plating was done in the face of obvious infection to prevent further cerebral herniation. Treatment with penicillin and sulfonamides was carried out uniformly.

In all patients debridement of scalp and brain tissue was carried out at the time of plating. During 3 operations (Cases 1, 5, and 6), large discrete cerebral abscesses were encountered and drained at the time the tantalum plate was introduced. In one instance (Case 2) primary scalp closure could not be accomplished at the time of cranioplasty.

In each case one or more attempts were made to obtain scalp closure over the plate following breakdown of portions of the wound. In 4 instances this included shifting of large pedicle grafts of scalp. These secondary interventions all failed, and all damaged additional areas of scalp, thus complicating final repair.

At the time of removal of these 8 tantalum plates thick infected granulation tissue was encountered both superficial and deep to the plates. Discrete collections of pus were found beneath the plates in 3 instances (Cases 5, 7, and 8). Wound cultures in all cases showed staphylococcus, of which 6 were *Staphylococcus aureus*. One wound contained additional marked pyocyaneus infection, and 2 contained streptococcus in addition to the staphylococcus. That invasion or extension by these organisms would have been difficult to control is indicated by the fact that in 3 of the 5 organisms tested for penicillin sensitivity, more than 10 units of penicillin per cc. were required to prevent growth.

DISCUSSION

Many reported cases do not give sufficient detail to determine the interval between secondary healing and later insertion of a tantalum plate. However, Woodhall and Spurling[7] reported plating in a case of frontal osteomyelitis and brain abscess within 3 months following aspiration and enucleation of the abscess; Robertson[5] performed tantalum cranioplasty $3\frac{1}{2}$ weeks after "removal of a brain abscess"; and Gardner[2] reported tantalum cranioplasty done at the time of drainage of a large cerebral abscess.*

The impression from these reports may be very misleading in indicating the absence of complications under conditions that we feel expose the patient to added risk. Until more adequate intervals of follow up have elapsed, such cases should not be presented as a basis for standard procedure.

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* The recent publication of W. J. Gardner (*Clev. Clin. Quart.*, April 1946, *13:* 72–87) recommends the use of tantalum for repair of cranial defects in infected cases and states that the tantalum implant does not impair healing in an infected wound. We feel that his 8 cases fail to prove his thesis. His 3rd and 7th cases died of deeper infection with the tantalum implants well-healed in place. His 5th and 6th cases survived to the writing of the paper after removal of the tantalum plates. In his 8th case the tantalum implant was tolerated in what proved later to be a tuberculous cerebral infection. It is our opinion also that his routine radical removal of a brain abscess wall is questionable.

Secondary intervention for tantalum plating in the presence of infection or immediately following wound healing cannot be justified on the basis of scattered successes. Before such a serious deviation from established surgical principles can be accepted as sound practice, it must be demonstrated that there is no increase in the risk of morbidity or mortality over more conservative management. We believe that the cases reported here emphasize the risks attendant on too early secondary intervention.

That late serious complications do occur following cranioplasty done under favorable conditions and with apparently adequate intervals after wound healing is evidenced by two cases mentioned by Woodhall and Cramer[6] in which brain abscesses occurred 8 and 10 months following tantalum repair.

The importance of covering a cranial defect has been overemphasized by some authors.[1,2,3] We do not feel that the defect itself is a cause of convulsive seizures nor that its repair will significantly alter the incidence of convulsions. This problem can best be approached with a discussion of cerebral hernia.

It is important to recognize that a cerebral hernia is an expression of seriously disturbed intracranial physiology and is not a disease *sui generis*. Following injury, cerebral herniation occurs as a result of one or several pathological processes. Extravasation of blood and edema are the early causes of cerebral herniation and must be dealt with surgically when necessary by evacuation of hematomas, appropriate hemostatic methods and debridement of devitalized tissue. Infection of cerebral tissue, with or without abscess formation, and frank meningitis are common causes of hernia after hours or days have elapsed. The remaining cause of serious cerebral herniation is interference with the absorption of cerebrospinal fluid by inflammatory reaction of the meninges, either due to actual infection or to the presence of blood or debris within the cerebrospinal fluid spaces. The use of the specific drugs, prophylactically and therapeutically, constitutes the most important phase of treatment. Spinal drainage both for infection and for blood in the cerebrospinal fluid is extremely helpful. Proper use of intraspinal penicillin in well tolerated amounts is very important in treating meningeal infections. Twenty thousand Oxford units of penicillin sodium diluted to 10 cc. with physiologic saline and barbitaged well through the spinal needle has produced no untoward reaction when used daily for as long as 10 days. The presence of actual abscess within or beneath the herniation must be suspected and explored for when indicated.

Treatment, therefore, should be directed toward the underlying causes of cerebral herniation and not toward the mere mechanical escape of brain through a cranial defect. In fact, the escape of damaged or infected cerebral tissue from a defect in the skull may permit survival in some patients when the maximal intracranial pressure compatible with survival had been approximated before herniation occurred. The preservation of cerebral tissue, however important to future motor or intellectual pursuits, is not essential to perfect preservation of the vital functions.

The decompression of non-localized cerebral space-taking lesions has properly fallen into disrepute amongst neurological surgeons. Nevertheless, it is important to recall that the maximal cerebral damage in penetrating head injuries (with the probable exception of through-and-through injuries) lies beneath the area of destruction of the calvarium. In this manner the cranial wound (which has often been debrided of bone fragments) acts as a decompression directly over the area of maximal damage. Herniation, in these cases, may permit exteriorization of a softened, hematoma-containing brain, or of a brain abscess.

Those who have suggested that cerebral herniation or degeneration results from the mere presence of a cranial defect have probably been influenced by the late pneumoencephalographic findings—the dilated ventricle beneath the defect and migration of both ventricles to that side. However, during the period of progressive herniation, entirely different relationships are usually present, with the ventricles displaced toward the side opposite the cranial defect. The ultimate contraction of devitalized tissue which occurs under favorable circumstances permits regression of the hernia and, if the destruction is great, traction upon the ventricular system follows.

In some instances, nevertheless, progressive ventricular dilatation occurs as a result of obstructive hydrocephalus or failure of the cerebrospinal fluid absorbtive mechanism, due usually to infection. Unless spinal drainage and heroic measures directed against infection control the process, a fatal outcome is inevitable.

If these untoward factors underlying cerebral hernia are controlled by appropriate treatment, recession of the hernia and wound healing result. After the wound is healed, it is our opinion that there is no emergency about covering the cranial defect.

It is of extreme importance not to procrastinate in the removal of a tantalum plate when infection and recession of wound margins indicate that ultimate removal is inevitable. Prompt removal of the plate and closure of the wound with provision for drainage and penicillin irrigation insure the success of the scalp operation and minimize the possibility of spread of infection to the cerebrum. With procrastination the fibrous contraction jeopardizes closure of the increasingly friable scalp and decreases the likelihood of subsequent successful repair in a single operation. Patient morale is sufficiently important to the surgeon for him to insist on gaining materially at every operative step.

EXPERIENCES WITH REPAIR OF CRANIAL DEFECTS BY TANTALUM

Of 44 patients whose defects we have covered with tantalum only one has required the removal of the plate. In this instance an extensive plastic procedure was necessary at the time of plating. A second attempt 10 months later was successful. Plates have been placed upon the outer table of the

defect margins and secured with tantalum triangles by using implements similar to those illustrated by Hemberger, Whitcomb and Woodhall.[4] The periosteum is sutured over the plate margins. Complete closure of galea and scalp is obtained by undermining and by other accepted plastic procedures. Drainage is not condoned, but postoperative aspiration may be necessary. Tantalum plates are formed before surgery by hammering the properly cut piece into the appropriately shaped spherical depression in an oak block with an oak hammer.

Four plates have been introduced at the time of primary operation, two in compound depressed fracture, one after excision of an osseous tumor and one to replace the anterior wall of the frontal sinuses after excision of an osteoma. Four plates were placed after cerebral scar resections, of which three included the ventricular wall. In the remaining 36 patients plating was performed with no accompanying intradural procedure.

Secondary plating has been delayed for a minimum of 3 months after primary healing, 5 months after delayed healing and 8 months after healing has been completed following a frank infection. Average intervals have been considerably greater. Systemic sulfadiazine and penicillin have been uniformly used immediately before and also following operation, but the specific drugs have rarely been used locally. Excellent cosmetic results have been obtained and the plate margins have not been palpable.

SUMMARY AND CONCLUSIONS

1. Tantalum may be safely used at primary operation to replace bone in a surgically clean procedure or in the repair of a compound depressed fracture if the contaminated wound has been undeniably converted to a clean one.

2. Secondary operation to cover a cranial defect should be delayed for a minimum period of 3 months after primary healing and of 8 months after healing if frank infection has been present.

3. The treatment of a patient with cerebral hernia should be directed toward the underlying pathological processes and not toward mechanical block of the escaping cerebral mass.

4. No clinical evidence of progressive deterioration of the cerebrum underlying a cranial defect has been observed after intracranial pressure has adjusted and complete epithelization has occurred.

5. Tantalum cannot be safely placed in an infected field.

6. Complete coverage with good scalp is essential to successful tantalum cranioplasty.

7. Unsuccessful tantalum plates must be removed promptly so that the patient at least obtains the benefit of excellent closure of the scalp and avoids the increased dangers of intracerebral infection.

8. Secondary covering of cranial defects with tantalum promises a high incidence of success if proper surgical principles are followed.

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OBSERVATIONS UPON THE MANAGEMENT OF ORBITO-CRANIAL WOUNDS

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PENETRATING cranial injuries encountered during war were frequently found to involve the fronto-orbital area of the skull. Among a group of 300 cases of compound fracture of the skull, there were 40 in which the orbit and brain were simultaneously injured. In 20 of these, one or both globes were anatomically damaged, producing blindness and requiring enucleation. Out of the experience in dealing with this type of wound at a General Hospital in the Theater of Operation, certain principles of procedure emerged pertaining to policies in management. These are briefly reviewed and illustrated.

MATERIAL

Two classifications were made of the 40 cases under consideration. Group I, numbering 20 patients, presented severe, destructive, anatomic injury to one or both globes in addition to the skull and brain injury. Group II, also 20 in number, comprised wounds which involved the orbit and brain with varying degrees of physiologic injury to the contents of the orbit.

In Group I, there were 16 cases with unilateral and 4 patients with bilateral globe destruction. One of these patients presented an associated transection of the cervical portion of the spinal cord. In 4 cases, the primary operation was performed at the 36th General Hospital. Five patients (31 per cent) required secondary debridement of the wounds due to incomplete primary operations in forward installations. Brain abscess occurred in 3 of the 5 patients. A frontal osteoplastic craniotomy flap was performed in 5 patients to debride adequately the brain and orbital injury. Massive wounds with extensive bone and soft tissue loss occurred in 5 cases.

Of the cases in Group II, 5 patients had primary debridement operations performed at the 36th General Hospital. Three patients (20 per cent) treated at Evacuation Hospitals, required secondary debridements, brain abscess being present in 2. Seven patients had total blindness of one eye; 8 had normal vision in the affected eye; 5 had vision which varied from light to finger perception.

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NEUROSURGICAL CONSIDERATIONS

General. The aim in the efficient care of this class of wounds would appear to be the achievement of primary wound healing following thorough debridement; the preservation of vision; the prevention of scar contracture; and the construction of the wound to permit the greatest conservation of tissue for subsequent reconstructive surgical procedures. This aim requires that the operative and postoperative care of the wound and the orbital

FIG. 1 (*left*). Example of a massive type of orbito-cranial wound.
FIG. 2 (*right*). The result of treatment of a massive wound by means of a temporary dural graft followed by a split-thickness graft to the granulated area.

contents be performed under as ideal conditions as possible. These conditions are present within a general hospital center. Five of the 16 cases (31 per cent) in Group I of both globe and brain injury required redebridement of the wounds, accountable mainly by the press of circumstances and the lack of time factors present in evacuation and field units. Survey of the patients with this type of wound indicated that they were safely transportable before operation in the great majority of instances. There is no contraindication to delay in operation upon the brain in the interest of the most adequate debridement. Similarly, enucleation of the globe may be reasonably delayed. Evacuation of the patient with this type of wound permits the forward surgeon to devote his time to the more urgent types of intracranial injuries.

The majority of the fronto-orbital wounds were observed to be satis-

factorily treated without complications by conventional methods of debridement through the wound of entrance. Certain particular features of the more complicated wounds will be briefly mentioned.

(1) *The Management of Massive Wounds.* There were 5 cases in Group I

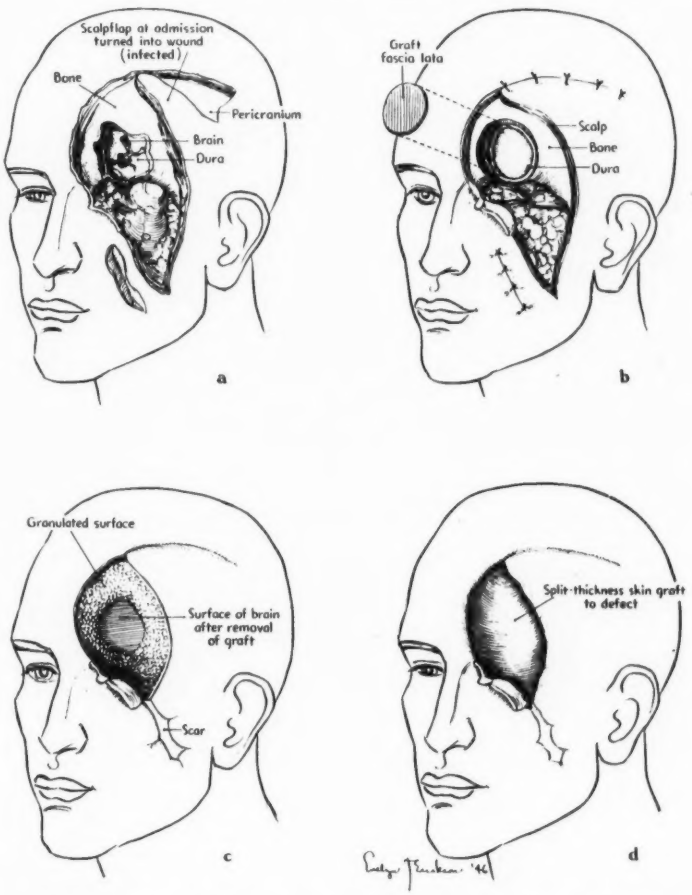

FIG. 3. Illustration of the technic employed in using a fascia lata graft to the evulsed dural defect following redebridement of the wound.

in which there was extensive bone and soft tissue destruction, as illustrated in Fig. 1.

The most suitable method in our experience, for accomplishing the aim in satisfactory healing of this massive type of orbito-frontal wound, was the use of a temporary fascial graft to the dural defect following debridement of the extra- and intracranial wound and enucleation of the globe. The para-

nasal sinuses require particular attention to insure adequacy in the removal of devitalized tissue, bone fragments, clot, and the sinus mucous membrane (Fig. 2).

This measure, as shown in Fig. 3, employs the use of a graft of fascia lata from the thigh to cover the evulsed dural defect in the massive wound. The graft is allowed to remain in place for 15 to 20 days, covered with vaseline gauze. By this means, spinal fluid leaking is controlled as well as cerebral herniation. At the end of that period, the graft is removed since it necroses in the open wound. The underlying brain and the surrounding granulating area are then prepared for a split-thickness graft by means of moist compresses of penicillin solution. The split-thickness graft may usually be applied a week after the fascial graft has been removed. Following this last procedure, the

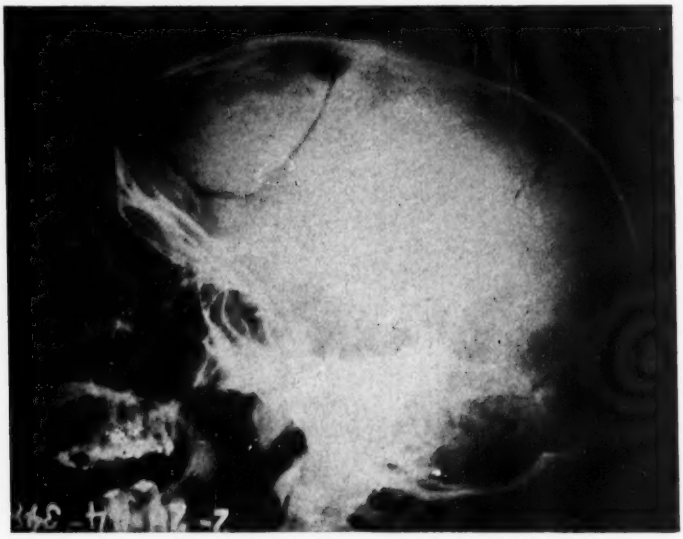

FIG. 4. Roentgen study of a wound producing globe destruction and penetration of the roof of the orbit and the frontal lobe. Debridement by means of a left frontal craniotomy.

patient is ready for evacuation to a plastic center for reconstructive surgery.

(2) *The Use of the Osteoplastic Flap.* An osteoplastic flap, used for exposure of the frontal fossa, was observed to be of value in the debridement of wounds with penetration produced by small metallic fragments entering the orbital roof and the frontal lobe at the base. The exposure provided in this manner permitted thorough debridement of the site of bony injury, the brain, and repair of the dura, either by suture or graft. The brain and cerebrospinal fluid pathways were thus isolated from the paranasal sinuses so frequently involved in the injury. The orbit may also be adequately viewed if necessary. By this means, a distortion of the frontal area through loss of

bone was avoided. The roentgen study in one patient thus treated is shown in Fig. 4.

(3) *The Treatment of Brain Abscess.* Five patients among both groups (3 in Group I and 2 in Group II) developed complicating brain abscesses (12.5 per cent). An incidence of 12.3 per cent was found to occur among 260 cases of penetrating cranial wounds of all types. One of the abscesses developed in a patient with an osteoplastic flap, pus being pocketed on the roof

FIGS. 5 and 6. Two cases of cranial wounds involving the orbit and frontal sinuses, complicated by brain abscesses and one by cerebrospinal rhinorrhea. Vision normal. Treatment by "open" method employing a temporary graft of fascia lata.

of the orbit, in a tract extending into the occipital lobe and also about a large ragged metallic fragment in the occipital area. Drainage of the anterior site of the infection was accomplished through the orbit, while drainage of the posterior area was performed following removal of the foreign body and insertion of a catheter into the tract which communicated with the frontal lobe.

In our experience, a complicating abscess in the frontal area communicating with the sinuses, with or without orbital involvement is, in general, most satisfactorily treated by an "open" rather than a "closed" method of management. The aim of wound healing in this type of situation requires the closure of the dura, thus isolating the brain from the sinuses. The use of a buried dural graft in an infected wound with overlying scalp closure, with the instillation of penicillin (by various technics), is not a secure method.

An "open" method used with success consisted in removal of the abscess by means of suction; closure of the dural defect with a graft of fascia lata; subsequent removal of this temporary graft, and the application of a split-thickness graft to the resulting defect (Figs. 5 and 6). This was similar to

the method used for the management of massive wounds with tissue loss and sinus involvement illustrated in Fig. 2.

ROENTGENOGRAPHIC CONSIDERATIONS

The roentgen examination of orbito-cranial wounds presents especially difficult technical problems. At the time of the initial examination, the condition of the patient may prevent the usual type of posturing for the frontal, Water's, or axial projections. In such instances, the head may be turned into the horizontal plane and projection of the beam at the usual angle carried out. Detail may be enhanced by the use of a wafer type grid in such cases. If the patient's condition permits, the studies may be made in the upright position, utilizing the vertical Potter-Bucky diaphragm. Bulky dressings should be removed, if possible, to minimize distortion. The routine examination should include, as a minimum, right and left lateral examinations, occipital, frontal, Water's axial or vertico-submental and detailed orbital films. The latter should be taken to visualize the optic foramina. Stereoscopic films in this instance may give additional detailed information and should be made if the patient's condition is satisfactory and the equipment suitable. The use of the smallest possible cone is recommended.

From a study of the films made in such manner, it should be possible to determine the extent of the fracture lines in the fronto-orbital area and visualize the pathway of a penetrating metallic fragment. Extension of the fracture lines into the frontal, ethmoidal, and sphenoidal sinuses or the optic foramina is significant preoperative information, as well as the visualization of depressed or indriven bone fragments.

OPHTHALMOLOGIC CONSIDERATIONS

The patients in Group I required the enucleation of one or both globes, a procedure which may be done to advantage before the cranial wound is attacked. The use of implants following enucleation depended upon the degree of disorganization of the orbital contents.

Twelve patients in Group I presented varying types and degrees of intraocular injury with complete or incomplete loss of vision. In 8 patients, the vision was normal. The predominant disturbance resulting in loss of vision was of vascular origin. The commonest findings were vitreous and retinal hemorrhages with tearing and detachment of the retina. Proptosis of the globe was frequently present with these lesions. In one patient, the attachment of the pulley of the superior oblique muscle was disrupted. In another, total blindness resulted from a fragment entering the roof of the orbit posteriorly from a parietal lobe site of entrance. Evaluation of the type and degree of damage to the globe and the optic nerve by either the fragment or the concussive effects of injury was difficult to make in the early period of management.

RESULTS

There were 5 deaths among both groups of patients, 3 being in Group I

and 2 in Group II, representing the mortality in a general hospital of 12.5 per cent. This figure is higher than that which was encountered at a general hospital in all groups with compound fracture of the skull (6.4 per cent in 260 cases—American). The orbito-cranial wound, however, in World War I was observed to be a hazardous type of injury involving, as it frequently does, the paranasal sinuses with the threat of intracranial infection.

Brain abscess occurred in 5 of the 31 patients who had primary treatment at evacuation hospitals. Four of these 5 complicating abscesses were successfully treated by means of the "open" method employing a temporary graft of fascia lata as previously described. The patient who expired was treated by an exteriorizing method without a graft (German POW) and subsequently developed a fungus.

Nine patients upon whom debridement was performed under ideal conditions in a general hospital did not develop inflammatory complications. The use of the temporary graft to isolate the brain from the sinuses was found to be a measure of considerable advantage for preventing infection in the "massive" wounds.

Two deaths occurred from conditions unassociated with the cranial injury, one from an accompanying transection of the cervical spinal cord and one following an orthopedic operation. A third patient died of infection following operation for brain abscess. The fourth death was that of a postoperative patient 24 hours after admission, due to massive intracranial injury. In a fifth, death occurred three days after primary debridement at the 36th General Hospital, the entire frontal area being evulsed by a gunshot injury.

Evaluation other than the mortality and the incidence of infection was difficult to determine in the short period (2–3 months) during which these patients were under observation. There was some indication that visual improvement occurred and was further possible in those patients with vascular lesions of the globe in Group II.

SUMMARY

1. A survey of 40 cases of orbito-cranial wounds was made in which 20 involved the globe, requiring enucleation, and 20 involved the bony orbit with varying degrees of physiologic injury to the globe.

2. In the achievement of ideal wound management it was held that this type of wound may be appropriately and safely transported to a general hospital for surgical treatment.

3. Massive wounds of the orbito-cranial area were satisfactorily managed by means of a temporary graft of fascia lata applied to the dural defect. After removal, the wound was grafted by means of a split-thickness graft and the patient was evacuated for reconstructive surgery.

4. An osteoplastic frontal craniotomy flap exposure was observed to be effective in dealing with wounds of this area in some cases.

5. Complicating brain abscesses were treated by an "open" method using

a graft of fascia lata to close the dura temporarily and isolate the brain from the sinuses. A split-thickness graft was applied to the wound after removing the temporary fascial graft.

6. Vascular lesions of the eye were noted in the majority of cases in which the globe was not disrupted.

7. The mortality in this type of injury at a general hospital was 12.5 per cent as compared with 6.4 per cent in penetrating cranial wounds in all areas of the brain.

EXPERIMENTS ON THROMBOSIS OF THE SUPERIOR LONGITUDINAL SINUS

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THE EXPERIMENTS to be described were planned to throw light upon the problem of "otitic hydrocephalus": a term originally introduced for a syndrome in which raised intracranial pressure and papilloedema are assumed to be due to sinus-thrombosis following otitis media, but are unaccompanied by evidence of leptomeningitis or of cerebral abscess. The cause and mechanism of this increased pressure are still controversial. Symonds (1937)[13] believes the syndrome to be dependent upon thrombosis: the lumen of the lateral sinus is blocked and the blockage extends across the torcular Herophili, thence ascending in some instances into the superior longitudinal sinus. Alternatively, in those cases where jugular compression proves both lateral sinuses to be patent, he suggests that the endothelium of the sinuses may be plastered with a thin layer of clot by retrograde extension from the lateral sinus to the superior longitudinal sinus, thus obstructing the arachnoid villi.

While the existence of the syndrome is generally accepted, the presence of an accompanying hydrocephalus is debated. According to some (Gardner, 1939[9]), the ventricles in these cases are of normal size or may even be reduced. Again, raised intracranial pressure with papilloedema may occur without clinical evidence of thrombosis or of antecedent infection either in the middle ear or elsewhere in the body—the so-called "pseudotumor cerebri" (Davidoff and Dyke, 1936;[7] Dandy, 1937;[5] McAlpine, 1937[11]). To such cases the term "toxic hydrocephalus" has also been given (McAlpine). No satisfactory explanation of the increased intracranial pressure has been offered for these. Yet there can be little doubt from certain records (Ellis, 1937;[8] Bailey and Hass, 1937;[1] Russell, 1944[12]) that thrombosis of the longitudinal sinus may be followed clinically by the syndrome and, in the cases quoted, ventriculography or postmortem examination has secured evidence of internal hydrocephalus.

In view of the existence of such cases it was felt that, could a spreading thrombosis from the longitudinal sinus be induced experimentally, the clinical and pathological study of such animals would throw further light upon the syndrome. So far as we are aware, only one attempt has been made in this direction. In one dog, Bize (1931)[3] occluded the torcular Herophili and hind end of the longitudinal sinus with a tampon of iodoform gauze, and in

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a second dog he injected 1 cc. of quinine urethane solution at the same site; in neither experiment was there any significant consecutive clinical or pathological change.

Since the middle segment of the longitudinal sinus appears to be the site of election in the initiation of spontaneous thrombosis (Welch, 1909;[14] Bailey and Hass, 1937[2]), this region was selected for our experiments. These fall into groups as follows:

A. *Rabbits*

1. *Inducement of Stasis.* The exposed sinus was occluded by a silver clip placed just anterior to the torcular Herophili (4 animals), or by two clips placed from 1.0 to 1.8 cm. apart (3 animals). The animals remained in normal health and were killed at periods from 2 days to 3 weeks later. None showed evidence of thrombosis either macroscopically or microscopically. The brains were normal.

2. *Stasis with Introduction of Coagulants.* The above procedure was combined with the injection of coagulants into the lumen of the sinus. (a) A physiological coagulant, thrombin, was used in 5 animals. When the greater part of the sinus had been exposed an occluding silk ligature was tied immediately anterior to the torcular, and about 0.1 c. cm. of a mixture in equal parts of a 10 per cent dilution of the thrombin solution and thorotrast was slowly (30 sec.) injected into the lumen anterior to the ligature. The mixture used was tested for us by Dr. R. G. MacFarlane and was found to clot rabbit's blood within 30 seconds *in vitro*. By adding thorotrast we hoped to gain radiographic information concerning the site of the expected thrombosis. The animals, however, remained normal and no trace of thrombosis was found on examination of the tissues from 5 to 22 days later.

(b) A chemical coagulant, ethamolin (an aqueous solution of 5 per cent ethanolamine oleate with 2 per cent benzyl alcohol), which is used clinically for the injection of varicose veins, was tried in 9 animals. In these experiments the ligature was omitted, and the lumen of the sinus was compressed in two places with artery-forceps while the injection was made into the segment between them. Both the forceps and the needle of the syringe were held *in situ* for 2 minutes after the injection had been completed. Most of the animals in this group appeared unduly excitable on the following day but, with two exceptions, they returned to normal and remained so until killed from 7 to 40 days later. The latter then showed neither thrombosis nor any other abnormality of the brain. Of the two exceptions one animal was found dead on the following day with extensive intraventricular haemorrhage, apparently due to accidental operative trauma. The other showed retraction of the head and convulsions following operation, and was killed on the second day, as it appeared moribund. The sinus at the site of injection and the great vein of Galen were greatly engorged, and there was moderate congestion of the superficial cerebral veins. The middle lobe of the cerebellum was swollen and protruded for a distance of about 0.6 cm. through the foramen magnum. On section the cortex forming the postero-medial angles of the occipital poles appeared softened and the occipital horns were dilated. On *microscopic examination* the torcular was partly occluded by recent thrombus but none was found in the longitudinal sinus. There was slight diffuse subarachnoid haemorrhage, more marked at the base of the brain than elsewhere. The occipital poles showed gross oedema of both grey and white matter, with occasional perivascular haemorrhages about the subependymal blood-vessels. The ependyma of the tips of the horns, which normally are not patent in the rabbit, was interrupted in many places by the blowing out of the cavity.

Comment. It was not expected that the degree of stasis induced by ligatures alone would effect thrombosis. But it seemed desirable to perform these initial experiments in view of the suggestion that the longitudinal sinus is particularly susceptible to thrombosis. But our failure when this measure

was combined with the use of coagulants was disappointing. Thrombin is a potent coagulant *in vitro*, and it had been anticipated that widespread thrombosis might preclude the survival of our animals. Ethamolin is an effective coagulant *in vivo* when used in the treatment of varicose veins but produced a limited thrombosis in one animal only, adjacent to, but not at the site of the injection.

To gain further information concerning the action of these two agents a series of experiments was then carried out upon the marginal vein of the rabbit's ear. In these it was found that, in parallel circumstances, the marginal vein was quite unaffected by the injection of undiluted samples of rabbit, bovine or human thrombin, but was invariably thrombosed, within two minutes of the injection, by ethamolin. The thrombosis occurred throughout the length of the compressed segment of the vein and proceeded to organisation during the succeeding 2 to 3 weeks.

The damaging effect of ethamolin upon the tissues was further demonstrated in 3 rabbits, by the application of the solution upon a pledget of lintine to the exposed cortex and leptomeninges. In two of the animals, killed 2 and 4 days later respectively, a wedge of haemorrhagic necrosis occupied the brain at the site of exposure. In the third animal, killed after 8 days, the cortex however appeared normal.

Since ethamolin appeared to be the most potent coagulating agent available, and since we had experienced some success in one of the nine rabbits in which it had been used, we decided to try it on the dog, where the sinus is of such dimensions that operative procedures can be better controlled than in the rabbit.

B. *Dogs*

1. *Injection of Ethamolin into Sinus* (Dogs 1, 2, 3). Under nembutal anaesthesia, supplemented when necessary with ether, the sinus was exposed over a length of 2 to 3 cm. Ligatures were tied to compress the lumen at either end of the exposed area and were released 2 minutes after 0.1 cc. of ethamolin had been injected into the intervening segment. Two of the animals were killed at the conclusion of the experiment (20 and 45 minutes after injection, respectively) and the third 2 months later. This last dog remained in normal health and activity during survival. None showed any abnormality in the dural sinuses or in the brain, either macroscopically or microscopically.

2. *Coagulation by Heat* (Dogs 4, 5). The sinus was exposed as before and the dorsal wall was cauterised, over segments measuring 0.4 and 1.5 cm. respectively, with a diathermy-point at a temperature just sufficient to coagulate muscle. In addition a ligature was tied at the posterior end of the exposed sinus in Dog 5. The animals showed no postoperative disturbance and were killed after 4 and 6 days respectively. Though no thrombosis was visible to the naked eye *microscopic examination* revealed a small mass of clot showing early organisation in the lateral angle of the sinus in the cauterised area in Dog 5. In both animals the wall of the sinus showed coagulative necrosis with diffuse extravasations of red corpuscles down to the intima, which was raised by oedema, the subendothelial spaces so formed being infiltrated with polymorphonuclear leucocytes. In both animals the brain was unaltered.

3. *Mechanical Blockage of the Lumen.* (a) *By muscle* (Dogs 6, 7). Portions of muscle, beaten out into small stamps such as are used by neurosurgeons to check haemorrhage, were introduced into the lumen of the sinus with the idea that the haemostatic action of the tissue

combined with mechanical obstruction might produce the desired effect. It was also anticipated that the necessary manipulations would cause some damage to the sinus endothelium. Therefore a small stamp, measuring 0.4 by 0.1 to 0.2 cm. thick in the case of Dog 6 and somewhat larger in Dog 7, was attached to a thread on a curved needle and was drawn after the needle into the lumen. Its entry was effected by enlarging the hole made by the needle with a sharp hook at the appropriate moment. Although ligatures had been temporarily placed on the sinus at either end of the exposed area the manoeuvre was accompanied by considerable haemorrhage which, however, instantly stopped when the muscle slipped into the lumen. The dogs remained well and active after operation and were killed after 4 and 10 weeks respectively.

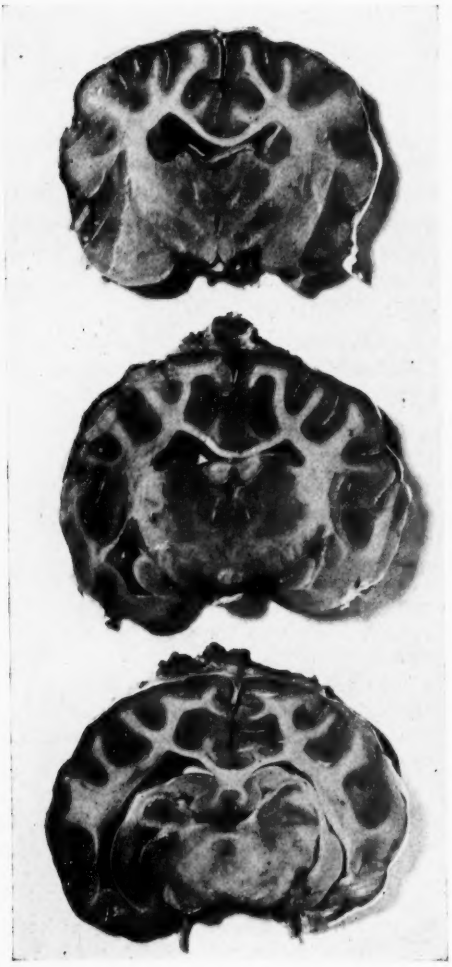

FIG. 1. Slices of brain from Dog 9, showing slight dilatation of left ventricle, and occlusion of longitudinal sinus in the two lower slices.

Macroscopic examination. In both animals the sinus appeared normal except where the muscle had been inserted. This was converted into firm greyish-white tissue which was firmly adherent to the dura, and at one level completely filled the lumen except for a narrow crescentic slit (Dog 6), or a pin-point lumen near one border (Dog 7). In both the brain appeared normal.

Microscopic examination. Transverse sections through the sinus showed replacement of the muscle by a circumscribed mass of fibrous granulation tissue, rich in young collagen fibres, enclosing islands of large mononuclear cells, many of which had formed foreign-body giant-cells about degenerated and atrophied muscle fibres. Throughout the tissue there were occasional spaces lined with endothelial cells and these, near the periphery, contained blood. Larger spaces, present between the sinus wall and the granulation tissue, appeared in part to be continuations of the lumen proper, and in part distended lacunae laterales.

Comment. Although the muscle had doubtless effectively blocked the lumen in early stages, it had become organised with considerable rapidity. Moreover there was no evidence that any thrombosis had been promoted by the muscle. The operation was therefore repeated using muscle that had previously been steeped in ethamolin.

(b) *Muscle steeped in ethamolin* (Dogs 8, 9, 10, 11). The procedure was similar to that in

(a) except that the muscle was steeped in ethamolin for about one hour before being introduced into the sinus. Increasingly large bits of muscle were used in successive experiments, up to 0.8 by 0.3 cm. in Dog 10 while, in Dog 11, three portions were separately introduced at different points into a segment of sinus about 3 cm. long. In Dog 9 the ligatures on the sinus were tied off for a period of 11 minutes, including 5 minutes after the muscle had been inserted. In Dog 10 (killed after 7 weeks) an attempt was also made to occlude the lateral sinuses by exposing them through a drill-hole made in the skull about 0.7 cm. lateral to the mid-line, and filling the hole firmly with bone-wax.

All the animals appeared in normal health until killed and showed no evidence of papilloedema. In Dog. 8, however, the wound opened after 9 days, following removal of the stitches, and discharged slightly; it was therefore killed on the 16th day. The remaining three were killed after 4, 6 and 8 weeks respectively.

FIG. 2. Transverse section of sinus in Dog 9, showing fibrous mass occupying centre of lumen, and crescentic spaces at periphery which were continuous with the main lumen at either end of the blocked segment. (Phosphotungstic-acid haematoxylin, ×22.)

On *macroscopic and microscopic examination* the appearances were similar to those obtained with the untreated muscle. In the dog killed after 16 days the sinus was blocked for a length of 1 cm. and the muscle was coated with a small amount of antemortem thrombus. But there was no further extension of the thrombus. In the remaining three animals a variable length of sinus, up to 2.5 cm. in Dog 9, appeared to be blocked by granulation tissue which, microscopically, was similar to that seen in (a). In spite of the addition of ethamolin the wall of the sinus appeared healthy. In Dog 10, in which the lateral sinuses were also attacked, the lumen of the right lateral sinus at the site of operation was compressed into a crescentic slit but was patent. Though the left lateral sinus had not been occluded by the bone-wax it was filled with recent thrombus which, however, on microscopic examination showed no evidence of organisation. The brains in all save Dog 9 appeared normal; in Dog 9 there was slight dilatation of the left lateral ventricle, most marked in the posterior horn (Fig. 1).

Comment. Obstruction of the lumen of the superior longitudinal sinus by fragments of fresh muscle, or by similar muscle steeped in ethamolin, was ineffective in promoting a spreading thrombosis. By the end of 16 days the muscle was replaced by granulation tissue and, at later stages, showed microscopic and sometimes macroscopic evidence of recanalisation (Fig. 2). The brain was unaffected with the doubtful exception of Dog 9, in which a maximal length of the sinus had been occluded, and the animals showed no clinical disturbance.

(c) *Cotton-wool* (Dogs 12, 13). Cotton-wool was substituted for muscle with the idea that this foreign substance might effectively occlude the sinus for a longer period of time. The wool was fashioned into an elongated sausage-shaped mass of suitable calibre and was intro-

duced into the sinus as before, after steeping it in ethamolin. Both animals remained healthy, with no evidence of papilloedema, and were killed after 10 and 7½ weeks respectively.

Macroscopic examination. In Dog 12 the sinus appeared to be completely obliterated throughout a segment, 2.2 cm. long, by tough grey fibrous tissue; the lumen elsewhere was patent and normal. The left lateral ventricle was slightly more capacious than the right anterior to the level of the occlusion but this inequality disappeared at more posterior levels (Fig. 3). In Dog 13 a length of 1.1 cm. was similarly obliterated. The sinus elsewhere was normal and the brain unaltered.

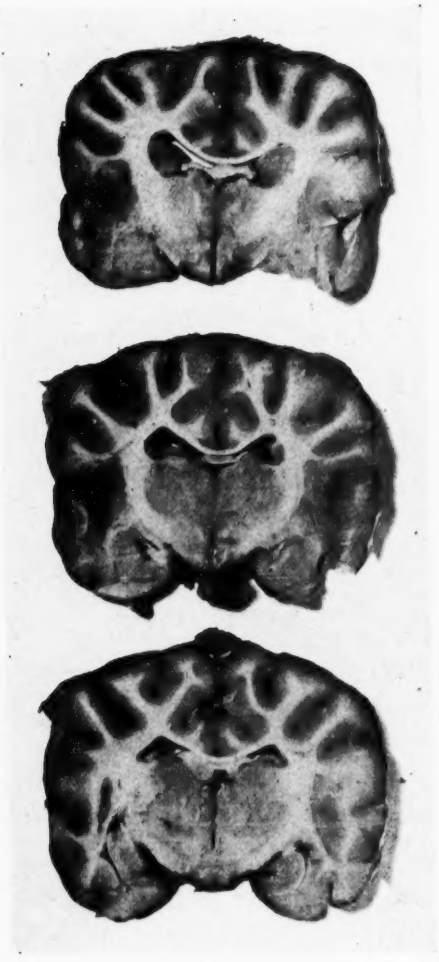

FIG. 3. Slices of brain in Dog 12, showing trivial dilatation of the left anterior horn. Note expansion of the longitudinal sinus by cotton-wool in lowest slice, with deformity of adjacent gyri.

Microscopic examination. The appearances in transverse sections through the occluded part of the sinus, were similar in both animals. The lumen was distended with fibrous granulation tissue which every where was closely applied to the wall of the sinus and separated the wool-fibres (Fig. 4). The fibres were frequently surrounded, wholly or in part, by foreign-body giant-cells. Numerous blood-vessels, mostly of small calibre and containing red corpuscles, were present in the granulation tissue (Fig. 5). The wall of the sinus was unaltered and the adjacent brain, apart from slight deformity caused by the pressure of the distended sinus, appeared normal.

Comment. The substitution of cotton-wool for muscle as a blocking agent made no difference to the results. It was observed, however, that in groups (b) and (c) a slight unilateral hydrocephalus had coincided with a maximal blockage of the sinus (Dog. 9, 2.5 cm.; Dog. 12, 2.2 cm.). In the experiments described an attempt was made to carry this observation further by blocking as great a length of the sinus as possible.

C. *Puppies and Kittens*

A litter of four puppies aged 6 to 7 weeks, and of four kittens aged 6 to 8 weeks were used because of the known high incidence of spontaneous sinus-thrombosis in young subjects, the advantageous dome-shaped skull of these young animals and the fact that the sutures of the vault were unclosed. The procedure was the same as in 3 (c) above. By using two elongated pledgets

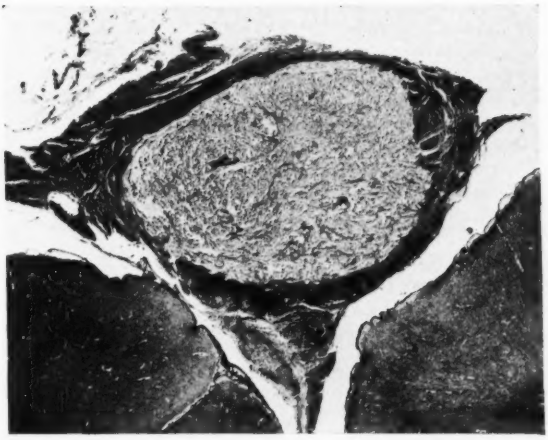

FIG. 4. Same as in Fig. 3, showing the sinus with occluding cotton-wool. (Phosphotungstic-acid haematoxylin, ×11.)

of ethamolinised wool, and passing these both forwards and backwards from a central point of entry on the fully exposed sinus, it was possible to obliterate lengths varying from 0.5 to 0.8 of the whole sinus (Fig. 6). With the exception of one puppy (3) all recovered well from this operation and remained perfectly normal until killed from 5 to 7 weeks later. There was no evidence of papilloedema. Puppy 3 did not regain consciousness and died after 36 hours, apparently from loss of blood combined perhaps with an excess of nembutal.

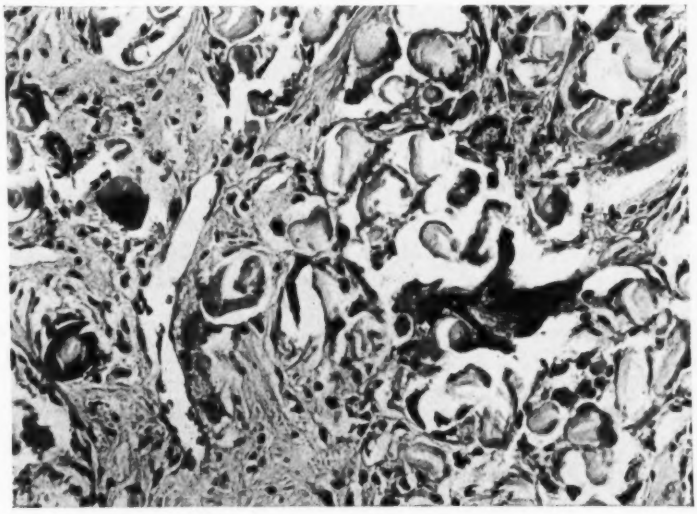

FIG. 5. Same as in Fig. 4, showing cotton-wool fibres (pale oval structures), foreign-body giant-cells, and a blood-vessel to left of centre. (Haematoxylin and eosin, ×270.)

In none of these animals was there thrombosis of the sinus either in front of or behind the blockage, which appeared complete throughout the greater part of the lumen. No abnormality was found in the brain in any of the kittens. In Puppy 3 the brain appeared full and soft and the convolutions were slightly flattened. There was uneven engorgement of the superficial veins. The wool filling the sinus was soaked with blood, and postmortem clot filled the residual parts of the sinus and the lateral sinuses. On section the cerebral tissue appeared soft and oedematous; the ventricles were small, their walls being in apposition. Of the remaining three puppies two (1 and 4) showed slight dilatation of both lateral ventricles, most marked in the body. In Puppy 2 the left ventricle was slightly more capacious than on the right.

Comment. Although it is possible, especially in puppies and kittens, to occlude the greater part of the longitudinal sinus, this operation is not followed by any unequivocal clinical or pathological evidence either of increased intracranial pressure or of hydrocephalus. The slight degree of dilatation observed in the puppies may have been significant; if so it is perhaps to be accounted for by the more numerous venous tributaries to the sinus found in dogs as compared with cats. But the dog has fewer tributaries than man and these experiments should be repeated on the monkey before any final decision is reached concerning the implications of extensive blockage of this sinus in man.

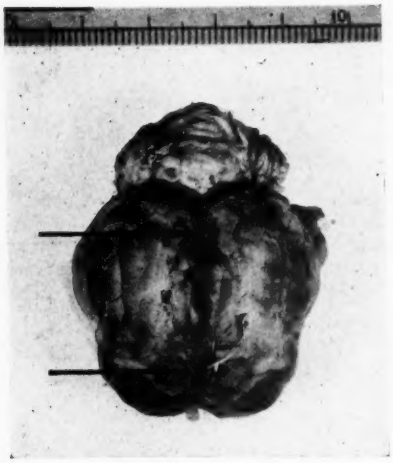

FIG. 6. Kitten 2. Dorsal aspect of brain with dura after fixation. The sinus has been occluded by cotton-wool between the points indicated by arrows.

The absence of any gross effects upon the brain in our experiments indicates that the collateral circulation is fully adequate to deal with the degree of disturbance that we were able to induce. It should however be noted that the anastomoses between the cerebral and diploic veins, demonstrated by Cushing (1902)[4] to be of great abundance and importance in the dog, were necessarily sacrificed in the operative exposure of the sinus in our animals.

It is possible that a condition analogous to that of "otitic hydrocephalus" in man can be produced experimentally only by inducing a thrombosis that will spread from the main sinus into the adjacent lacunae and the mouths of the venous tributaries. Reviewing the published reports of human cases of this kind one is struck by the frequency with which an infective process is present in some part of the body although the thrombosis of the intracranial sinuses is essentially aseptic. It may be—and the idea is not new—that tissue-immunity is a factor deserving of more attention in this connection. With this in mind we attempted to reproduce the Shwartzmann phenomenon in the walls of the longitudinal sinus in rabbits as follows:

D. *Shwartzmann Phenomenon in Rabbits*

1. In two rabbits the centre of the sinus was exposed over a length of about 1 cm., and its dorsal wall was well scarified with the point of a needle. A pledget of cotton-wool soaked in a tested potent filtrate of B. coli, kindly prepared for us by Dr. E. S. Duthie, was laid over the area for 10 minutes and the wound was then closed. Twenty-four hours later a precipitating dose of 4 c. cm. of the filtrate was injected into the marginal vein of the ear. An hour later both rabbits appeared prostrated; respiration was rapid and they had passed a large quantity of fluid faeces. On the following day they appeared more lively and, two days after the injection, their behaviour was normal. They were killed 3 and 9 days respectively after the operation.

Macroscopic examination. In the animal killed after 3 days there was haemorrhage and necrosis of the soft tissues bordering the operation area. The dura however appeared normal and showed no thrombosis of the sinus. The brain also was normal. In the animal killed after 9 days there was no haemorrhage in the tissues and the gap in the skull was occupied by yellowish-grey granulation tissue. Beneath this the dura and brain appeared normal; there was no thrombosis.

2. On the supposition that the application of the filtrate to the sinus had been inadequate we repeated the experiments on 4 rabbits. The dura over the sinus was scarified more vigorously and, after applying the filtrate on cotton-wool as before, the bony defect was filled with a layer of stiff agar mixed with the filtrate in equal parts. The wound was closed leaving this mixture *in situ*. In one animal the dura to one side of the sinus was accidentally torn during the manipulations.

The behaviour of these animals after the precipitating dose was much as in (1) but the scalp, in a broad zone about the wound, became haemorrhagic and proceeded to dry gangrene in two examples. When sacrificed 2, 6, 9 and 17 days respectively after the operation the macroscopic changes at the site of operation were more intense than in (1), especially in those with necrosis of the scalp. In these (killed after 6 and 9 days) the dura was overlaid with opaque yellow fibrinoid material without evidence of granulation tissue. Nevertheless the dura itself was unaltered and the sinus was patent. In the animal in which the dura had been torn a triangular area of haemorrhage, 0.9 by 0.4 cm. and 0.4 cm. deep, occupied the subjacent cerebrum.

Microscopic examination. Residual portions of agar embedded in purulent necrotic debris overlaid the dura. Occasionally the whole thickness of the dura was infiltrated with polymorphonuclear leucocytes. Yet the lining of the sinus was in all instances unaltered and the lumen was empty. The haemorrhagic area found in the cerebrum in one rabbit, beneath a tear in the dura, showed ill-defined necrosis of the cortex and white matter. Many vessels in both the leptomeninges and brain were necrotic and were surrounded by haemorrhage. The meninges were moderately infiltrated with leucocytes. There was no definite thrombosis in the cerebral veins.

DISCUSSION AND SUMMARY

Though we failed in our objective in these experiments the results are instructive in various respects. The idea that thrombosis may readily be induced in the superior longitudinal sinus evidently needs revision. Though the blood-flow may be sluggish, and the walls rigid, thrombosis will not follow occlusion even when combined with the use of irritant coagulants such as ethamolin. But ethamolin with temporary compression will invariably induce thrombosis in the peripheral vein of the rabbit. The physiological coagulant, thrombin, proved another disappointment: although the samples used were potent *in vitro* they exercised no perceptible effect *in vivo*. We can offer no explanation of this anomaly.

It was remarkable to find that, in the dog and cat, the greater part of the sinus could be suddenly obliterated without any obvious impairment of normal function on recovery from the anaesthetic. Evidently the collateral circulation can at once compensate for such a disturbance. And reparative processes, ending in organisation and recanalisation of the occluding material, whether this be muscle or cotton-wool, proved surprisingly effective. It is possible that a condition analogous to the "otitic hydrocephalus" of man can only be achieved experimentally by inducing a thrombosis that will extend from the main sinus into the adjacent lacunae and the mouths of the venous tributaries. We did not succeed in this in any type of experiment. In support of this theory are the published reports (Dandy, 1940;[6] Jaeger, 1942[10]) of resection of considerable lengths of the longitudinal sinus in the course of operation for the removal of tumours without subsequent clinical disturbances referable to this measure.

A spreading thrombosis such as we visualise may be impossible without the presence of infection providing a humoral factor such as we invoked in our experiments with the Shwartzmann reaction. In these experiments we obtained necrosis of all tissues that were exposed to the filtrate with the exception of the dural walls of the sinus. A conspicuous reaction was evoked in the brain in one animal in which the dura was torn. The remarkable immunity of the dura is doubtless correlated with its relative avascularity, for the Shwartzmann phenomenon appears to be essentially a vascular mechanism. We believe that the problem of producing the syndrome of "otitic hydrocephalus" experimentally lies along these lines, and it is hoped that our records may provide some guide in the planning of any future attempts in the direction.

It is a pleasure to thank Professor J. H. Burn and the staff of the Department of Pharmacology, Oxford, where most of this work was carried out. Special grants for expenses were allowed by the Medical Research Council, to whom our thanks are also offered.

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CASE REPORT AND TECHNICAL NOTES

EPENDYMOMA OF SPINAL CORD: SUBTOTAL INTRAMEDULLARY REMOVAL FROM SYRINGOMYELIC CAVITY

CASE REPORT

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(Received for publication February 7, 1946)

The following case is reported primarily to illustrate the value of intramedullary exploration of the spinal cord, particularly where the primary diagnosis is difficult to establish. (For several months prior to operation the case was considered as an example of conversion hysteria.) It is also of interest that a syringomyelic cavity was associated with the tumor; and that after operation the patient suffered "tract" pain of spinal cord origin.

HISTORY

Chief Complaint. Weakness of legs and sensation of pressure in girdle distribution following herniorrhaphy under spinal anesthesia. Symptoms were of approximately 2 years' duration.

Present Illness. This 35-year-old soldier first noted an uncomfortable pressure sensation in July 1943, immediately after the repair of a right inguinal hernia under spinal anesthesia. The entire abdominal wall was affected, from the level of the 12th ribs to the groins bilaterally. In addition he complained of vague numbness and weakness in the legs. These symptoms lasted about 2 weeks and did not recur until February 1945. At the time of recurrence the patient was overseas and his outfit was alerted for a move forward. No definite neurological signs being found, he was thought to be suffering from conversion hysteria.

Prior to herniorrhaphy he had noted pain of an entirely different character from that of his present illness. There were no known complications during the spinal anesthesia.

Past History. Civilian occupation: Boilermaker. Tropical service: None. Habits: Very moderate use of tobacco, alcohol; no drugs. Illnesses: Usual childhood diseases with no known sequelae. Venereal disease denied. Injuries: Struck in occiput early in 1943, after which he was unconscious for about 10 minutes. The only residual symptoms were occasional mild dizziness and slight blurring of vision on reading. Operations: Herniorrhaphy, inguinal, spinal anesthesia, July 1943.

Family History. No known mental or nervous diseases.

Course of Present Illness. In February 1945 the patient awoke one morning unable to turn in bed because of pain in the lower thoracic spine, accompanied by numbness and weakness of the legs. The back pain was accentuated by attempting to turn, and was accompanied by the same girdle-like sense of uncomfortable pressure that he had experienced nearly 2 years previously. Coughing or straining never aggravated his symptoms, though change of position, particularly forward bending, did. The initial acuity of these complaints soon abated, though the symptoms persisted to the time of operation, along with a tendency to staggering gait, and occasional "dancing" (clonus) of the feet. Tingling sensations of glove distribution began to affect the hands and forearms in February 1945.

Because of these complaints he was admitted to a General Hospital overseas, where the following notes were made: 13 March 1945, normal reflexes of lower extremities; no Babinski. Lumbar puncture: Initial pressure 215 mm. CSF; no block; total protein 27 mg. per cent. X-rays of spine showed minimal osteoarthritic changes in the lower dorsal and upper lumbar vertebrae. 27 March, tests with a Richter neurodermometer showed no sensory loss, which

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together with the history and vague neurological findings led to a tentative diagnosis of conversion hysteria.

On 28 March 1945 the tendon reflexes of the left arm were found slightly more active than those of the right; there was a positive left Hoffmann reflex; the lower abdominal reflexes were sluggish; and there was hypalgesia from the nipple line downwards, with equivocal impairment of position sense and reduced vibratory perception in the toes of both feet. He walked with a rather wide base. On 1 April the sensory level was found to be at the um-

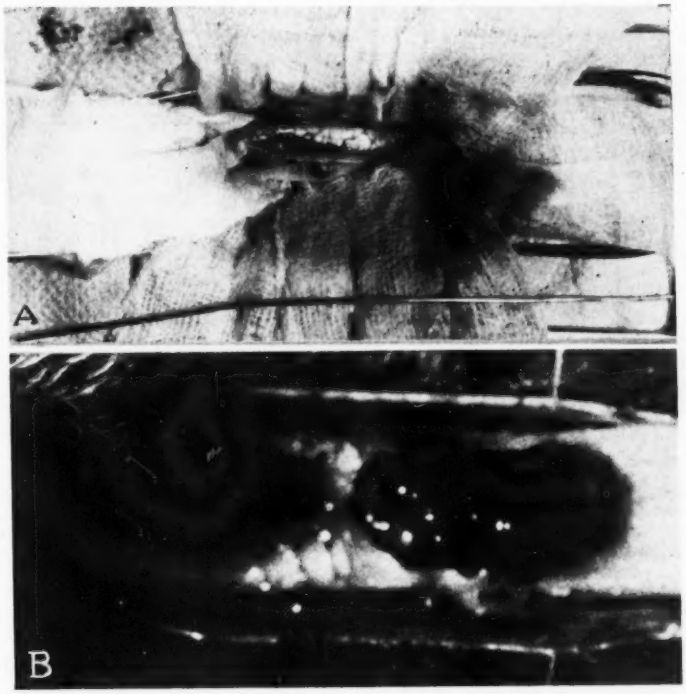

FIG. 1. Photographs of (A) the spinal cord, Th. 8–Th. 10, before removal of tumor, and (B) the tumor tissue (ependymoma), immediately after intramedullary removal, placed on the cottonoid strip at the right. The operative opening in the dorsal part of the cord appears at the left.

bilicus. The diagnosis of pernicious anemia was considered until the gastric analysis and blood counts proved to be normal. By 15 April there was a suggestive left Babinski and bilateral ankle clonus.

On 10 July 1945 he was admitted to a General Hospital in the United States with the same complaints. Initial neurological examination disclosed fibrillations of the muscles of the shoulder girdle; some spasticity of the lower extremities; a wide base, some staggering of gait, and a slightly positive Romberg test. The tendon reflexes of all four extremities were hyperactive and rated as 3; the upper abdominals were listed as 2; the lower abdominals 0; cremasterics 2; Babinski positive bilaterally; Hoffmann negative.

Sensation: Glove distribution of hypesthesia almost up to the elbows; variable and vague bilateral sensory level in region of umbilicus downwards for pain, with preservation of touch. Vibratory and thermal perception reduced from knees downwards; position sense

reported intact. There was no evidence of neurofibromatosis. Lumbar puncture: "Slightly xanthochromic" cerebrospinal fluid; total protein 25 mg. per cent. Grant-Cone test: Slightly positive. Laboratory work-up: Within normal limits. Serology: Negative. Tentative diagnoses: Conversion hysteria; or amyotrophic lateral sclerosis secondary to spinal anesthesia.

Myelography. This revealed an almost complete central block at the level of the 10th thoracic vertebra. The patient was transferred to Neurosurgery for operation.

Laminectomy, Th. 8–Th. 10 inclusive. Anesthesia: Intratracheal gas-oxygen-ether. The spinal cord was swollen for a distance of 2.5 cm., almost completely filling the intrathecal space. Its dorsal surface appeared somewhat irregular and blanched, and palpation suggested the presence of an intramedullary cyst, confirmed by midline aspiration of the distended cord with a fine hypodermic needle, which yielded a few drops of yellow fluid. The dorsal aspect of the cord was, therefore, split along the midline for 2.0 cm., disclosing a soft mass of irregular reddish-blue tissue 2.0 cm. in length and 1.0 cm. in diameter. The bulk of this tumor was carefully shelled out of a smooth lined cystic cavity in the center of the cord (Fig. 1). At the ventral aspect of the cyst, fine strands of tumor tissue extended into the substance of the cord. These were not molested lest viable cord be damaged. It was noted that the cystic cavity, cephalic end, was funnel-shaped, suggesting the prolongation of a syringomyelic cyst rather than a degenerative area caused by pressure ischemia.

Microscopical Diagnosis. Ependymoma.

Postoperative Course. Eight hours after operation the patient was able to move both legs to some extent. There was almost complete anesthesia for all modalities from Th.10 downwards, and urinary retention. The patient complained of a continuous tingling burning pain that seemed to affect both lower extremities in diffuse fashion, and was different from any sensation he had noted prior to operation. This sensation gradually disappeared as his control of bladder and legs improved. Three weeks after operation, he began to walk and had regained approximately 75 per cent pain and position sense in the left leg, and 50 per cent pain perception on the right but no position or vibratory sensation. The level of hypesthesia was Th. 8 on the right and Th. 10 on the left, with approximately 50 per cent sparing in the saddle area. Voluntary bladder function had returned to normal. The numbness of hands and forearms completely vanished after the operation.

On the 21st postoperative day, the patient was discharged to another hospital for deep x-ray therapy, in excellent condition, with strength and sensation improving steadily.

COMMENT

With regard to the incidence and distribution of spinal cord ependymomas, Shenkin and Alpers[4] state that 10–20 per cent of all spinal cord tumors are intramedullary gliomas, of which ependymomas are the predominant type, and that the highest incidence of ependymomas is within the thoracic segments. In 7 of their 27 reported cases there were associated cystic cavities of the cord. Associated sensory disturbances suggesting "tract pain" are characterized by diffuse painful sensations of burning or boring character, distant from the tumor level, and neither of radicular nor peripheral distribution. Intramedullary removal of ependymomas is not a novel procedure.[1,2]

Differential diagnosis, according to most writers on the subject of intramedullary cord neoplasms, constitutes an acknowledged difficulty. For example, Lichtenstein[3] speaks of remissions reminiscent of multiple sclerosis, and emphasizes the fact that multiple intramedullary tumors of the cord sometimes occur, making diagnosis still more difficult.

The present case report illustrates many of the features common to reports in the literature; e.g. (1) the difficulty of diagnosis, illustrated by the fact that conversion hysteria, pernicious anemia and amyotrophic lateral sclerosis were seriously considered; (2) the thoracic site of the tumor; (3) the associated syrinx; and (4) the possibility of a multiple process, suggested by the fact that the "glove" hypesthesia and paresthesia vanished after operation. Perhaps a syringomyelic cavity of the cervical cord may have been drained through the central canal when the cord was opened at the thoracic level.

The difficulty of early diagnosis in this and similar cases should carry a note of warning, especially since remissions may occur during the development of neurological symptoms and signs which suggest demyelinizing diseases of the spinal cord.

As to prognosis, ependymomas are usually slowly growing tumors, and do not tend to metastasize. They are, however, resistant to x-ray therapy, and hence the ultimate prognosis should be guarded.

The painful dysesthesias noted by this patient and others mentioned in the literature are apparently of spinal cord origin. This is worthy of note since there seems to be some controversy as to whether such a phenomenon exists. However, the pain experienced postoperatively in the present instance is similar to the burning dysesthesias felt "in the legs" of some paraplegic patients with traumatically divided spinal cords, and suggests abnormal stimulation of sensory pathways either by a pathological or physiological process, acting within the cord itself. Since effective pathological, physiological or operative interruption of the spinothalamic pathways too often fails to relieve this type of pain, such dysesthesia may well be due to abnormal neural volleys reaching the thalamus via other sensory pathways; i.e., via the intact portions of the dorsal columns immediately above the site of the cord lesion. These tracts may be stimulated pathologically by cicatricial traction or by changes in the local fluid or vascular milieu; or neurogenically, by disturbed sensory circuits.

SUMMARY

1. A case of intramedullary spinal cord tumor (ependymoma) at Th. 8–Th. 10 is reported.
2. Syringomyelic cavitation (perhaps multiple) was associated with the tumor.
3. Subtotal surgical removal of the tumor was accomplished.
4. Pitfalls of diagnosis are briefly discussed.
5. The significance of pain of intramedullary spinal cord origin is mentioned.

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METHOD OF VENTRICULAR FLUID REPLACEMENT FOLLOWING VENTRICULOGRAPHY

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(Received for publication January 28, 1946)

A description of a method designed to replace ventricular fluid following ventriculography is presented. Replacement of ventricular fluid is often desirable in hydrocephalus of congenital origin, but more especially in those cases due to a periaqueductal stenosis. Attempts to lessen reactions, for example, by the use of oxygen instead of ordinary air, during and following ventriculography, are well recognized. Other methods for replacement of ventricular fluid also have been utilized.

MATERIALS NECESSARY

The ordinary equipment for ventriculography is required. In addition a 500 cc. pyrex washing bottle* with glass fitting stopper, as illustrated (Fig. 4) and 5 feet of plastic or rubber tubing are needed. One end of tubing should be fitted with a rough glass adapter to provide tight fitting with the ventricular needle (Fig. 5).

PREPARATION OF EQUIPMENT

The pyrex washing bottle is chemically cleaned with a standard "bichromate-sulfuric acid" solution.

The plastic tubing may be chemically cleaned, depending on the type of plastic material used. Rubber tubing should be washed thoroughly and soaked in distilled water overnight.

After equipment has been carefully chemically cleaned it is then autoclaved for 30 minutes at 20 pounds pressure and 260°F. and then wrapped sterilely.

PREPARATION OF PATIENT AND VENTRICULOGRAPHY

Following the skin preparation, two posterior parietal twist drill or burr holes are inserted on either side of the midline. In extreme hydrocephalus in children whose cranial sutures have separated, two posterior parietal 1 cm. skin incisions may be all that is necessary. With the patient on the side, the ventricular needles (Fig. 5) are then inserted into both lateral ventricles (Fig. 1). The lower needle is connected with the flask at the intake as shown (Fig. 4),

FIG. 1. Under local anesthesia 2 ventricular needles are inserted into the ventricles. To the upper needle is attached a two-way stop-cock, and to the lower needle is attached the chemically clean sterile plastic tubing, connecting the ventricular system with the flask.

and the oxygen is inserted into the upper ventricle by very light pressure (Fig. 2). A constant check should be made to insure an even stream of fluid from the lower needle. The chin may be rotated upward near the end of the procedure to facilitate a more complete drainage of the ventricles. The needles are removed, and the connection with the flask clamped. The flask and tubing are then wrapped in sterile covers and kept at room temperature while the patient is taken to the x-ray department for the ventriculographic series.

* May be purchased from Fisher Scientific Co., Pittsburgh, Pa., Catalogue No. 3-406.

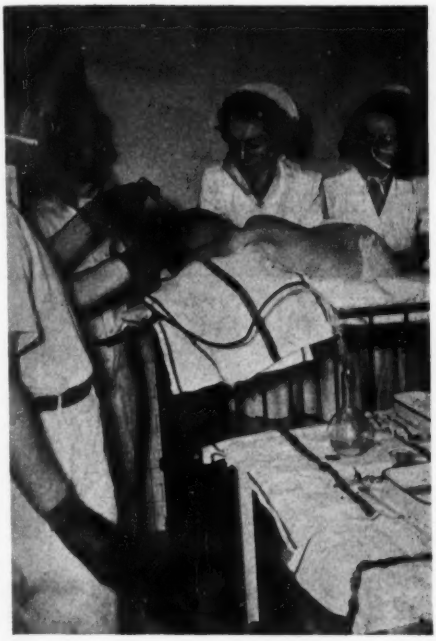

FIG. 2. Oxygen is then inserted under light pressure into the upper ventricle while the ventricular fluid flows into the flask below head level. The chin may be slowly rotated upward near the end of the procedure to more completely empty the ventricular system. Both needles are removed and the patient is taken to the x-ray department for the ventriculogram series. The flask and tubing are wrapped sterilely and kept at room temperature until x-ray studies are completed.

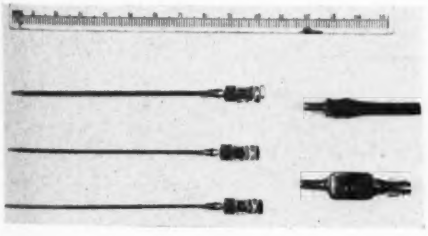

FIG. 5. Showing from above: the No. 18 hollow biopsy needle, the No. 20 hollow biopsy needle and the ventricular needle. Each are modifications of the standard lumbar puncture needles. At the right above is the rubber adapter for the brain needles, and below is the adaptable suction tip used largely for fine cortical resections.

FIG. 3. The patient is returned to the dressing room on the ward and the needles are re-inserted. The upper needle is now left open while the flask is raised to allow the fluid to be replaced. The oxygen thus escapes through the upper needle. Note the amount of fluid (500 cc.) being replaced here. Case: 5-year-old girl with stenosis of aqueduct.

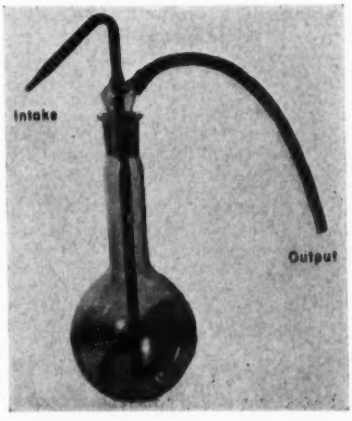

FIG. 4. This 500 cc. pyrex washing bottle is used for receiving and storing of the ventricular fluid.

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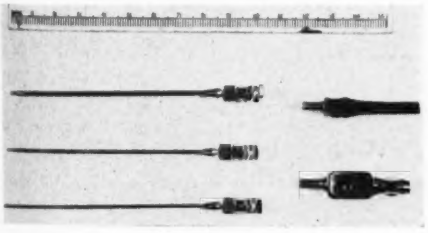

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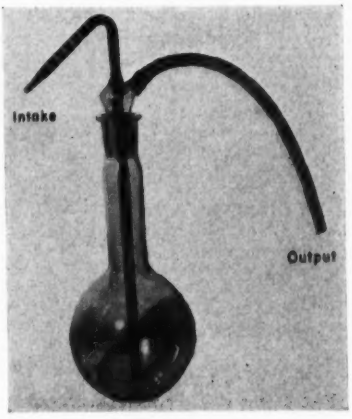

FIG. 4. This 500 cc. pyrex washing bottle is used for receiving and storing of the ventricular fluid.

The patient is then returned to the dressing room or operating room following x-ray studies, and the skin re-prepared. With the needles in the ventricles again, the tubing is transferred to the output (Fig. 4) and connected with the lower ventricular needle. The stilette is then removed from the upper ventricular needle while the flask is inverted and elevated above the level of the patient's head (Fig. 3). The oxygen will then escape through the upper needle while the fluid is replaced through the lower one. The height of the bottle should be approximately 12 inches above the lower ventricular needle, so that the replacement of ventricular fluid will be gradual.

SUMMARY

A method of replacing ventricular fluid following ventriculography is reported.

Emphasis should be placed upon thorough chemical cleaning of the flask and proper sterilization. The possibility for reactions is thus minimized.

This method has been used repeatedly in this clinic with satisfaction and without reactions to the procedure of ventriculography in cases of extreme hydrocephalus.

A REMOVABLE SUTURE METHOD OF NERVE REPAIR

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(Received for publication February 9, 1946)

A successful nerve repair requires a minimum of fibrosis at the site of suture. All present day technique is directed towards the prevention of such fibrosis by careful hemostasis, the utilization of impermeable sleeves, and the use of non-irritating sutures.

The placing of an epineurial suture, even with the utmost care, often results in the piercing of the perineurium and superficial fascicles by the point of the needle. The result is scarring, augmented if a suture remains in the area. There is a cellular infiltration about the most inert of our present day sutures, tantalum.

A method of nerve anastomosis minimizing trauma to neural tissue and permitting of subsequent removal of all suture material would seem to be important in combating fibrosis. The method described in this report is a composite of suggestions submitted by the members of a neurosurgical section familiar with nerve repair. The basic idea is taken from the tendon "pull-out" suture of Dr. Sterling Bunnell.

Two repairs have been done by this method. The first has been reexplored and is now available for evaluation.

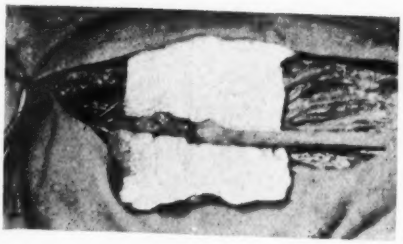
FIG. 1. Gross pathology of the injury. There is an hourglass constriction of the nerve with neuromatous tissue proximally. Microscopic sections taken from this level show fibrous continuity only, with a few regenerating neuraxes penetrating the stroma. In all photographs the proximal nerve is at the left.

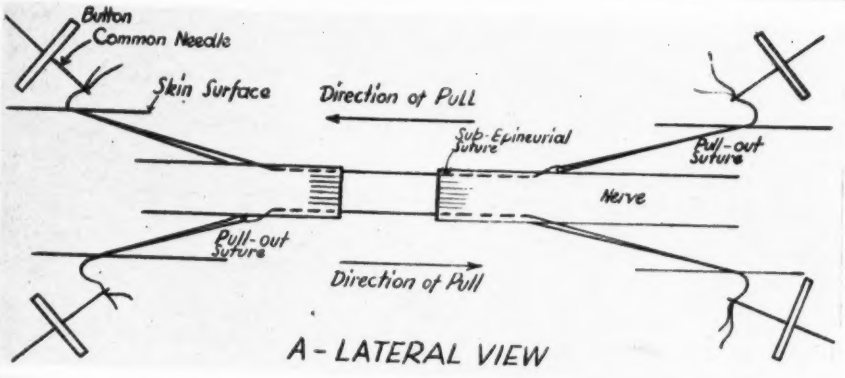
FIG. 2. Anastomosis completed. The tension sutures have been brought through the skin and secured over the buttons. The pull-out sutures have yet to be brought to the skin surface. Note that tension is in the axis of the nerve.

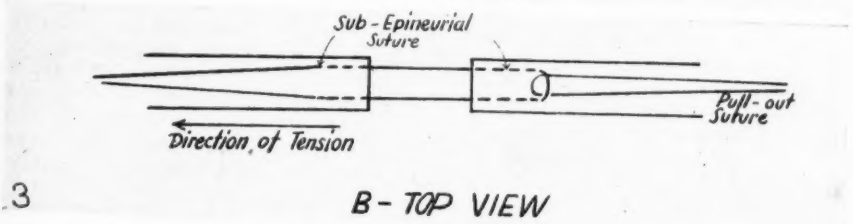
FIG. 3. Diagrammatic representation of the suture.

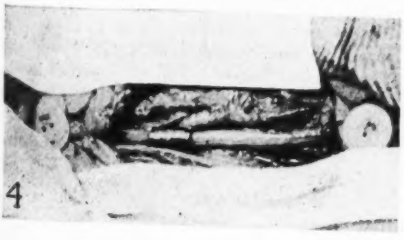
FIG. 4. The wound prior to closure. A tantalum scroll encloses the anastomosis and wires.

FIG. 5. Anastomotic site 57 days after suture. The foil has been removed to show the neo-membrane.









FIGS. 1–5. See opposite page for description.

CASE 1

D.M. was wounded in action on 4 Jan. 1944, suffering a penetrating rifle bullet wound of the left arm. The missile entered the anterolateral aspect of the arm, emerging on the posterolateral surface. There was no concomitant bone or vascular damage. Immediate numbness of the lateral surface of the forearm, and of the thumb, forefinger, and middle finger occurred.

Upon examination at this hospital on 26 Feb. 1945, the wounds were healed following a secondary closure overseas on 21 Jan. 1945. There was a "hot spot" over the neurovascular bundle in the arm at the level of injury. Light percussion produced a burning pain projected to the area of anesthesia, which included the thumb, forefinger, and middle finger. No voluntary contraction could be elicited in those muscles supplied by the median nerve.

On 8 March 1945, the left median nerve was explored under infiltration anesthesia. The pathology is demonstrated in Fig. 1. Direct stimulation above and below the lesion by faradic current evoked no motor response. Some sensation could be appreciated below the lesion; this was interpreted as being a partial regeneration of a few pain fibers rather than a sparing of pain elements. Serial sections of the nerve were taken until satisfactory tubules were encountered; the defect at this time measured 45 mm. Mobilization of the nerve was then carried out over a sufficient distance to permit the approximation of the ends without tension.

Two .005 gauge tantalum tension sutures were then utilized as shown in Fig. 3. These were threaded between the epineurium and perineurium by grasping the wire in a hemostat, after the ends were bevelled with the scissors to facilitate passage. When done in this manner there was minimal damage to the fascicles. The wires were then threaded on a common needle and brought through the subcutaneous tissues and the skin in the axis of the nerve at a sufficient distance to preclude upward angulation of the anastomotic site.

The pull-out wires were then placed and brought to the skin surface on a common needle. By traction upon the tension sutures the nerve ends were brought into apposition; these sutures were then tied over buttons, securing the anastomosis. Two marking sutures of .003 tantalum wire were then placed in the epineurium near the approximated nerve ends. These sutures will appear as markers on subsequent x-rays, and their separation would identify a disruption when stretching of the nerve is begun after removal of the pull-out sutures.

Fig. 2 illustrates the anastomosis prior to the application of the tantalum sleeve (Fig. 4).

The wound was then closed in layers by fine silk sutures and a posterior plaster splint applied to maintain 90° elbow flexion and relaxation of the anastomosis.

The postoperative course was uneventful. Some postoperative discomfort was anticipated because of motion of the tension sutures with the skin. This was not the case; contraction of the biceps or triceps did not produce pain or paresthesias.

Three weeks after operation, on 29 March 1945, the tension sutures were divided at the skin after the usual antiseptic precautions. They were then easily withdrawn by traction on the pull-out wires. The patient experienced transitory paresthesias, but no pain. The sites of emergence of the sutures in the skin were closed when examined 2 days later.

Extension of the elbow was begun in 3 weeks after x-rays were taken to determine the distance separating the marker sutures. These films showed the tantalum sleeve to have become partially unrolled. The patient showed a progressive, rapid advancement in Tinel's sign, which finally reached the mid forearm. On 28 April 1945, the muscles of the forearm were surveyed by faradic stimulation over the motor points. No contraction of the pronator teres or other muscles supplied by the median nerve at lower levels could be elicited.

On 4 May 1945, the anastomotic site was reexplored for removal of the displaced foil and direct stimulation of the nerve. Exposure was under infiltration anesthesia. After removal of the foil a satisfactory neo-membrane was seen to cover the anastomosis except at the posteromedial aspect, where the tantalum had slipped; here there were adhesions of the nerve to the adjacent tissues. When this area was mobilized, the patient complained of local pain, probably the result of escaped fibers. The site showed no other marked evidence of neuroma formation, except for increased firmness to palpation (Fig. 5).

The nerve was uncovered for a distance of 70 mm. beyond the suture site. With stimulation of 3 volts at this level, the patient appreciated pain projected to the index and middle fingers. No pronator function was elicited. Stimulation with ½ volt produced pain just distal to the suture but not at any distance below this level. It was concluded that a satisfactory downgrowth of pain fibers had taken place, an excess of 70 mm. in 57 days, and that there was no indication to further expose the nerve to obtain the lower limits of regeneration.

At the time of this exploration the elbow was in 135° of extension, and from the photograph it is obvious that the nerve was under no tension (Fig. 5).

The patient was then given further physical therapy directed toward complete extension of the elbow joint; however, he did not receive daily galvanic stimulation of the denervated muscle groups, which perhaps would have been desirable. The Tinel's sign showed a continued advancement, during which period the patient was given a series of convalescent furloughs.

On 13 Sept. 1945, 6 months after operation, the Tinel's sign had advanced to a point 18 in. beyond the zone of suture. This advanced averaged 3 in. a month, or 2.4 mm. a day, and sensory return had progressed as far distally as the proximal phalanx of the indicus. Motor evaluation on this date showed return of function of the pronator teres, the flexor carpi radialis, and the flexor pollicus longus. There was equivocal return of function of the sublimis tendon to the index finger; no function of the thenar group of muscles could be elicited.

Comment. The rate of advance of the Tinel's sign in this case, 3 in. a month, kept abreast of but did not exceed the average rate of regeneration in comparable cases done at this hospital by the conventional methods.

The writer wishes to emphasize that this case was selected. The satisfactory regeneration was dependent upon these considerations primarily: a relatively brief time elapse since injury, and the ability to appose excellent nerve ends without tension.

The follow-up study in the second case done by this method has also been completed.

CASE 2

This patient suffered a complete division of the left ulnar nerve immediately proximal to the olecranon groove. The transection proved to be approximately 2 cm. above the motor branches to the flexor carpi ulnaris muscle. A suture by the pull-out technique was performed after transplantation of the nerve. This was done on 4 April 1945, 4 months after injury; the defect in the nerve prior to transplantation measured 2.4 cm.

The pull-out sutures were removed in 3 weeks, and the postoperative plaster, which maintained elbow flexion, was removed in 5 weeks.

There was progressive advancement of Tinel's sign, which had reached the hypothenar eminence on 1 Sept. 1945; concomitantly there was perceptible contraction of the flexor carpi ulnaris.

Upon final examination, on 4 Dec. 1945, there was early return of the first dorsal interosseus; none of the remaining intrinsic muscles supplied by the ulnar nerve were functioning. Superficial sensation had returned to the tip of the 5th finger, as had pain to pin prick and deep sensation. The phenomenon of axone splitting was present.

The patient has subsequently been separated from the service.

CONCLUSION

This type of nerve anastomosis may have a place in cases selected for a suitable site of injury and for the ability to approximate nerve ends without tension. A removable suture should be of particular benefit in those fortunate cases in which there is minimal intrafascicular and perineural fibrosis.

TWIST DRILL GUARD FOR USE IN CRANIOTOMY

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In the course of work performed on experimental animals it became necessary to drill holes in the cranium. To obviate puncture damage of brain substance when using simple

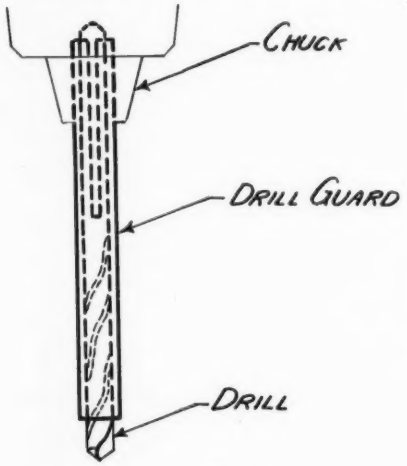

FIG. 1.

twist drills, a circumferential drill guard was developed.* The simplicity and general applicability of the drill guard (or stop) for use with ordinary twist drills, for craniotomies, prompts this presentation.

The drill guard consists of a slotted cylindrical tube that surrounds the drill (Fig. 1). Circumferential pressure on the drill guard by the drill chuck maintains the drill at the desired cutting length.

It has been found that for best results the cylinder should be drilled with the size drill that is to be used in the operative work. With monel metal a 3/16 inch inside diameter cylinder need have a wall thickness of only 1/32 of an inch. The slot may be cut with a thin jeweler's saw or even with a fine toothed hacksaw. When the drill guard fits the drill snugly, only moderate pressure of the chuck jaws is necessary to insure fixation of the drill during operation.

* The idea of this device evolved during a conversation with Mr. Jack Bronson, Machinist at the Naval Medical Research Institute. The instrument is now made by the J. Sklar Manufacturing Company, 38–04 Woodside Avenue, Long Island City, New York.

RADICAL SURGERY AND PENICILLIN IN BRAIN ABSCESS

A METHOD OF TREATMENT IN ONE STAGE WITH SPECIAL REFERENCE TO THE CURE OF THREE THORACOGENIC CASES

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(Received for publication March 20, 1946)

IN MOST instances the bacteria responsible for brain abscesses are sensitive to penicillin. For this reason a considerable improvement in the prognosis of these lesions is to be assumed. In this, our experience undoubtedly agrees with that of the majority of neurosurgeons. There is, however, a tendency toward the belief that the use of penicillin may either simplify the surgical treatment of brain abscesses or actually do away with such treatment. On the contrary, we believe that, thanks to penicillin, more radical operations can be undertaken successfully for abscesses which otherwise could not have been cured. The method that we advocate, and that we have used in the treatment of every brain abscess encountered during the past fifteen months, may be summed up thus: Complete extirpation of the abscess, no matter what its form, whether acute or chronic, single or multiple, even though this may mean opening up a communication between the abscess and the ventricle.

We have operated upon 17 brain abscesses in this way. In the first 3 cases, when penicillin was not available and when complete extirpation could not be carried out, the patients did not survive. In the following 14 cases (13 with penicillin and 1 without) the patients were all completely and rapidly cured. It should be added that 10 of these 14 cases showed a serious form of abscess, including 3 which were metastatic from the lungs.

We came to adopt the complete extirpation in one stage by utilizing in general the method described by Clovis Vincent,[8] with the addition of penicillin. Vincent's method of dealing with both acute and chronic abscesses may be summarized as follows:

1. *Acute abscesses.* In order to lower the threatening increased intracranial pressure, the abscess is first tapped. A bone flap is then turned down in front of the abscess without opening the dura. This usually suffices to lower pressure sufficiently so that further procedures may be postponed until the abscess has formed a capsule.

2. *Chronic abscesses.* This means, as a rule, well encapsulated abscesses. They should be extirpated "en masse," without opening the capsule and without drainage.

While the complete extirpation method proved to be a great improvement in many instances, it was still inadequate in dealing with certain types of abscess, as Vincent pointed out himself.[9] These types were:

1. Acute abscesses in which inflammatory brain oedema and temporal herniation below the tentorium are such that a large decompressive flap is not enough to prevent death from intracranial pressure and disturbance of the brain stem.

2. Acute abscess so near the meninges that a decompression would be likely to cause rupture of the abscess into the subarachnoid space.

3. Chronic abscess too large or too deep to be taken out "en masse" without great risk of damage to the basal ganglia and brain stem.

4. Chronic abscess without well defined capsule. Extirpation "en masse" for this type of abscess would almost certainly involve opening the abscess and often contamination of the ventricle with serious risk of meningitis.

With the advent of penicillin a reduction in the incidence of postoperative meningitis was to be expected. This led us to adopt in all cases of brain abscess, irrespective of type, a much more radical procedure than before. Since the aim was to cure the abscess by a one-stage operation it was felt that the procedure should be complete extirpation even if this involved opening both the abscess and the ventricle.[6] In acute abscesses the chief point is to reduce the temporal herniation. For this purpose, tapping the abscess is usually wholly inadequate. We therefore turn a flap, open the dura and remove not only the abscessed zone but also a large area of contiguous inflammatory and oedematous white matter. This is similar to the operation for an infiltrating glioma. In chronic abscess the important thing is to take out all the capsule walls, however thick or thin they may be, as well as all the abscesses which may be multiple, and in addition, all the fibrous formations so frequently found in the vicinity of abscesses, as these are often the origin of recurrences. Complete extirpation of a large abscess without endangering the brain stem can only be accomplished after deliberately opening the abscess or even taking it out in fragments in a manner similar to that used when dealing with a large basilar meningioma.

In short, we feel more and more that with the use of penicillin, brain abscesses should be dealt with like brain tumors. On the other hand, if fairly simple abscesses are met with, simpler surgery might be sufficient, such as tapping in the case of a mild acute early abscess. As a neurosurgeon I see very few of these.

Let me now give, as examples, the reports of three cases of thoracogenic brain abscesses. One case was acute and the other two were chronic. After these case reports, further details as to how we deal with various forms of brain abscesses will be given.

CASE REPORTS

Case 1. Acute thoracogenic brain abscess.

Lag., male, aged 32, was in good health until 1945. Mar. 30, 1945: Rhinopharyngitis followed by pneumonia. Apr. 26: Pneumococcic pleural empyema. May 1: Pleurotomy and drainage (Dr. Moulonguet). Sulfa drugs given for 2 weeks. Improvement. May 30: X-ray still showed opacity with air level. Reoperation. Improvement. July 30: Suppuration started again and continued in spite of treatment by sulfa drugs for several weeks. Sept. 27: Pleurot-

omy, lower than at first operation. Oct. 7: Epileptic fit and sudden right hemiplegia with aphasia. Brain abscess suspected. Oct. 11: General treatment by penicillin started.

Admission to la Pitié, Oct. 20, 1945. The patient was in a state of drowsiness, with relaxation of sphincters and stiffness of neck. Temperature was normal. He was aphasic: sometimes understood simple questions but could only nod in answer. There was right complete hemiplegia: arm hypertonic; leg hypotonic. Periosteal reflexes were increased, except for the ankle jerk, which was weaker. Babinski was positive. There were right hypoesthesia, difficult to elicit, and right facial weakness. The pupils were dilated. Fundi and visual fields were normal.

Course. Oct. 22, 1945: Coma. Slow breathing. Oct. 23: Left occipital abscess found by burr hole. Abscess tapped and 20,000 units of penicillin injected locally. Slight improvement for some hours. Then deterioration. Only a few drops of pus yielded on repeated tapping. Pus contained gram-positive cocci looking more like strepto- than pneumococci. Culture negative.

Operation. Complete extirpation of abscess (acute). On Oct. 25, 1945 a left occipitotemporal flap was made. The dura was tied to the pericranium by catgut sutures, and the flap protected by several layers of gauze. By tapping through the unopened dura the lesion was localized in the lower occipitotemporal region. The dura was then widely opened, exposing an abscess the size of a small hen's egg. Dissection was fairly easy in the occipital lobe, where the abscess had a thin capsule. In the temporal lobe, however, there was no well defined capsule, but fibrous and soft tissue from which pus exuded. In this deeper and diffuse part, a branch of the abscess extended far into the temporal lobe (Fig. 1). Finally the abscess with its overlying pia was completely excised. With a view to reducing intracranial pressure and temporal herniation, about half as much of oedematous white matter of the brain was removed. The ventricle was not opened, but the subarachnoidal space communicated with the bed of the abscess, where 20,000 units of penicillin were instillated. The dura was tightly closed and flap replaced. Closure without drainage, with 20,000 units of penicillin between flap and scalp. Bacteriological examination of pus: same as above.

Postoperative Course. The patient's condition immediately improved. For 4 days: Penicillin, 100,000 units, intramuscularly and intravenously. For 2 days: Penicillin, 20,000 units, between flap and scalp. For 1 day: Penicillin, 10,000 units, by lumbar puncture; on this day (Oct. 27, 1945) cerebrospinal fluid contained 21 white cells/mm. 3. Blood count: 4,185,000 red cells; 7,200 white cells with only 60 per cent polys. Bacteriological examination of wound fluids never showed any bacteria. The patient's condition improved steadily and after 3 days he was completely conscious. His aphasia became less marked and on Oct. 30, 1945 he could understand and execute simple orders. There was now right hemiparesis instead of hemiplegia. He had a right transitory hemianopsia.

Nov. 5, 1945: Apart from slight headaches his condition was even better. No hemianopsia. Wound completely healed. Nov. 18: Patient normal except for difficulty in reading and weakness of right shoulder muscles. No headaches. On Nov. 29, 1945 he was discharged.

The patient subsequently developed pulmonary congestion in the left lung, which lasted less than a week. When last seen, on Jan. 15, 1946, his temperature was normal and the right pleural sinus had been closed for several weeks. X-ray examination confirmed the probability of cure.

As far as the condition of the brain is concerned, there was no sign of recurrence. The fundi were normal. The abscess had been completely extirpated, and it was felt that a ventriculogram was unnecessary to confirm the cure. The flap and occipitotemporal scar were normal. It may be noted that the patient never had a fit since operation.

Case 2. Chronic thoracogenic brain abscess.

Bou., male, aged 46, was in good health until 1942.

June 1942: Gangrenous abscess of lung. Incomplete recovery after 2 months. May 1945: Recurrence of acute pulmonary symptoms with foul sputum. Treated by bronchial aspiration. Continued to produce more than 400 cc. sputum daily. Aug. 15: While the patient was sitting in the garden of the hospital there was sudden onset of dizziness, headache, and weakness of

left limbs. After 2 to 3 days his condition improved. Aug. 25: Recurrence of left hemiplegia, which became spastic the following day. Headaches in frontal and occipital regions with feeling of great weight. No vomiting. No fits. No stiffness of the neck. Temperature near to normal.

Admission to la Pitié, Sept. 5, 1945. Examination Sept. 7: The patient was in poor general

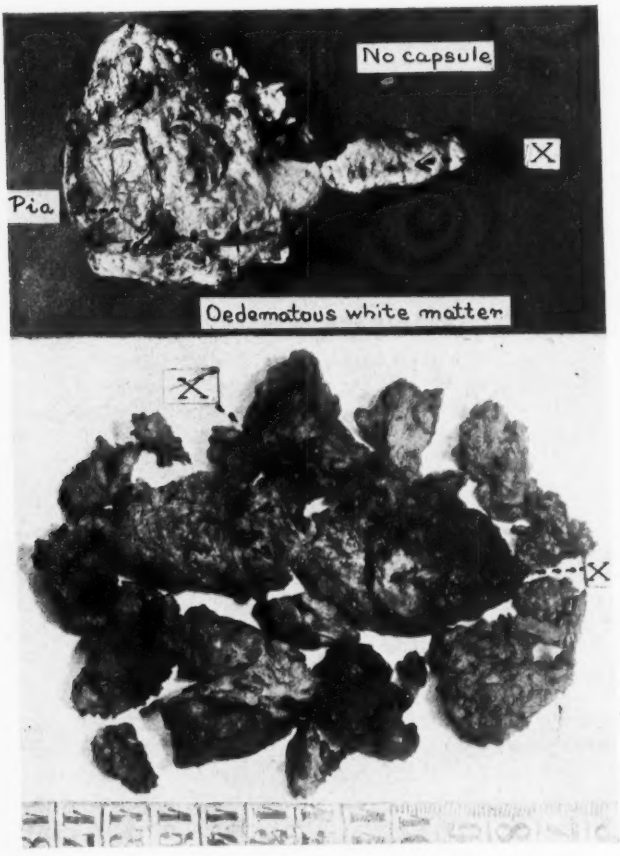

FIG. 1 (*above*). Case 1. Acute left temporo-occipital abscess. (X) Fibrous formation.

FIG. 2 (*below*). Showing fairly typical appearance of an irregular abscess removed piecemeal, as in Case 2. (X) Fibrous formation.

condition, conscious, and without signs of intracranial pressure. He had a typical left spastic hemiplegia with left facial weakness, and diffuse left hemi-hypoesthesia. No stiffness of the neck. There was slight bilateral papilloedema. Visual fields were normal.

Operation. Complete extirpation of abscess (chronic). On Sept. 18, 1945, through a right temporal burr hole, an abscess was found which had no definite capsule near the body of the ventricle. It contained foul pus, and since the organisms were regarded as probably anaerobic

and not likely to be responsive to penicillin, it was felt that a bone flap would become infected. A semicircular incision in the scalp was therefore made in the occipitotemporal region, and the temporal muscle incised. The burr hole was then enlarged by rongeurs to a diameter of 6 cm. Dura and cerebral cortex were opened with a diathermic needle, and the abscess was found at a depth of 4 cm. It was the size of an orange and had no well defined capsule, but contained pockets of foul pus, some limited by thick walls, some by thin fibrous formations. In places, there were fragments of capsule. It was out of the question to perform an extirpation "en masse" without opening the abscess or abscesses, and therefore a piecemeal removal was made by aspiration and diathermy (Fig. 2). Every suspect area was excised, leaving a clean parieto-temporo-occipital cavity about 10 cm. deep and 3 cm. wide. The ventricle was not seen, but deep in the occipital lobe, near the falx, fragments of capsule led to the tentorium and subarachnoidal spaces. Penicillin (10,000 units) together with V.187* (2 grams) was instilled into the cavity. The wound was closed in layers without drainage, with 5,000 units of penicillin and 2 grams of V.187 between dura and muscle.

Bacteriological Examination. The pus contained gram-positive cocci. Culture: After 3 days pneumococci and long hair-like gram-negative organisms (probably B. trichoides).

Postoperative Course. Patient received for 4 days 80,000 units of penicillin intravenously and intramuscularly, and 20,000 under the scalp. For 4 more days, 12 grams of methyldiazine were given orally. There was never ground for fear of meningitis. No organisms were found in the wound and none were grown. After 2 weeks the patient was nearly cured. Examination Oct. 5, 1945: Consciousness normal; no headaches; still slight weakness of left limbs. The visual fields were normal but there was still papilloedema. This gradually subsided. He was discharged from the hospital on Oct. 27, 1945.

On Feb. 1, 1946 the patient was reexamined. There was no sign of recurrence in the brain. But although a period of improvement followed penicillin therapy, the lung abscess was not completely cured, and a surgical intervention was considered. It may be pointed out that gram-positive cocci, gram-negative bacilli, and long hair-like gram-negative organisms were found in the sputum. Thus in the brain and in the lung there was association of at least two organisms responsible for the abscesses, aerobic and anaerobic.

Case 3. Chronic thoracogenic brain abscess.

Cat., male, aged 46, was in good health until 1942.

Aug. 1942: Operation for left inguinal hernia. One week later signs of right pulmonary infection soon followed by purulent sputum. In spite of lipiodol, bronchoscopy, and sulfamidotherapy, x-rays showed persistently a typical picture of abscess of the right inferior lobe. For 3 years there were remissions and recurrences of clinical signs. Nov. 1945: Patient admitted to Hospice de Bicetre with hyperthermia, pain in right side of chest, and purulent sputum containing pneumo- and staphylococci. Improvement for a few days. Surgical treatment of lung abscess was advised. Jan. 15, 1946: Patient began to complain of inability to read newspapers; he had to read several times to understand the meaning. This grew progressively worse. Jan. 2: Headaches started in frontal region and became increasingly severe. Feb. 5: Patient said he could no longer see on the right. Feb. 14: Cerebrospinal fluid tap: 22 lymphocytes and 0.85 alb.

Admission to la Pitié. Feb. 16, 1946. Examination showed a thin and slightly dyspneic man, who had to sit up to breathe freely. Consciousness good. No stiffness of neck. No Kernig sign. Blood pressure 150/100. No fever. There was no gross aphasia, but he did not understand complicated orders and made mistakes in simple arithmetic tests. Alexia was absolute: the patient could not name even isolated letters or recognize the same letter in different words. He could write his name or a simple phrase. There was gross disturbance of memory. Motility: Left side normal; right arm normal; slight weakness of right leg with brisk ankle jerk and questionable Babinski sign. Other reflexes normal. Sensibility: Super-

* V.187 is Methyl p-amidino-phenyl-sulphone hydrochloride. It was found experimentally to be very active against anaerobic organisms.

ficial, normal; deep, grossly disturbed in both right limbs. No cerebellar signs. Cranial nerves: Normal except for right hemianopia, bilateral moderate papilloedema and slight hypoexcitability of left vestibular nerve. Diagnosis: Left occipital abscess.

Penicillin (100,000 units) was given intravenously and intramuscularly during the 24 hours before operation.

Operation. Complete extirpation of abscess (chronic). On Feb. 19, 1946, through a left occipital burr hole, the abscess was found at once. About 15 cc. of foul pus were aspirated. Penicillin (20,000 units) was instilled in the abscess cavity and iodine put in the burr hole.

An occipital bone-flap was then carried out. The dura was very tense and on opening it there was marked herniation of the brain. The abscess, about the size of a hen's egg, was

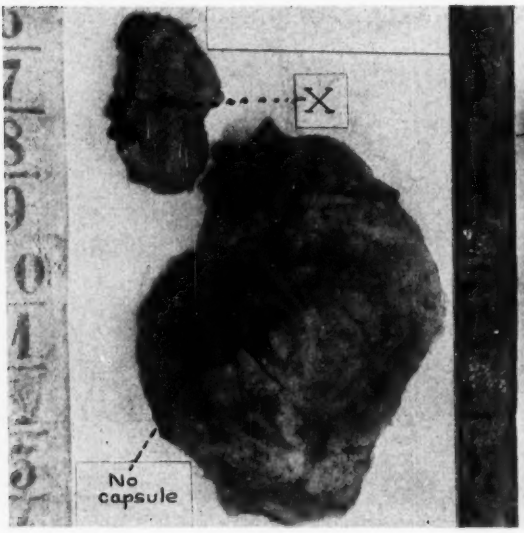

FIG. 3 (*left*). Case 3. Chronic left occipital irregular abscess. (X) Fibrous formation.
FIG. 4 (*right*). Chronic right prefrontal abscess of regular type which followed an ethmoidal fracture.

situated in the postero-internal part of the occipital lobe. It was quite superficial near the midline, and after removal of some fluid oedematous white matter it showed a tendency to extrude itself. Its capsule was well formed anteriorly and externally, but very thin internally, where, under the pia, there was an open area about 4 cm. square. After removal of the abscess, a smaller fibrous mass was disclosed near the midline, and this was excised (Fig. 3). No other abscess was felt by blunt needle probing. The ventricle was 1–2 mm. from the external limit of the abscess, and 10,000 units of penicillin were instilled into the ventricle, and a paste of 15,000 units and 2 grams of V.187 was spread into the cerebral bed of the abscess. The dura was tightly closed, and the flap replaced and covered with the same dose of penicillin-V.187 paste as above. Closure of muscles and skin in two layers.

Bacteriological Examination. The pus contained gram-positive cocci and gram-negative bacilli with hair-like gram-negative threads very similar to those in Case 2. Cultures: No growth.

Postoperative Course. This was uneventful, with no clinical or bacteriological signs of any

infection. Patient received daily 200,000 units of penicillin and 12 grams of methyldiazine for 4 days; locally, 20,000 units of penicillin daily between flap and scalp for 4 days.

Feb. 21, 1946: Sputum showed staphylococci and a few gram-positive bacilli. Feb. 26: Cerebrospinal fluid, 13 lymphocytes, 0.56 alb. Patient was up 3 weeks later. To make sure of recovery a ventriculogram was done Mar. 11, 1945 and showed normal ventricles. Mar. 12: Patient knew letters and started reading again. Mar. 13: Right hemianopia. Slight right papilloedema. Patient was sent back to his former hospital with advice to have his lung abscess operated upon.

COMMENT

Complete extirpation was performed in all 3 cases of thoracogenic abscesses, 1 acute and 2 chronic, the latter belonging, as will be shown later, to the "irregular" class. The abscesses were all opened during the operation, and their contents came in contact with cerebrospinal fluid from either the ventricles or the subarachnoid space. No signs of postoperative infection appeared, and indeed, the patients were able to be up and leave the hospital in about a month. It is interesting to note that in all three cases, the abscess was in the occipital lobe, whatever the reason may have been.

In Cases 1 and 2, 5 months and 6 months respectively have elapsed since operation, and complete cure is very likely. In Case 3, which is more recent, a ventriculogram was normal, and this is probably the best presumptive evidence of complete cure. No fits have been observed to date.

The very serious prognosis of thoracogenic brain abscess is well known (Cairns,[1] Pennybacker,[7] Cohen and Drooz[2]). However, with complete extirpation and the use of penicillin, we have not observed any significant difference in the postoperative course of brain abscesses, regardless of their origin. In 14 consecutive cases the abscesses were cured, and of these, 4 were due to craniocerebral wounds, 1 to a fracture of the ethmoid, 3 to a meningitis, 1 (cerebellar) to otitis, 2 were metastatic from an infection of the face or neck, and 3 were thoracogenic. It is noteworthy that no postotitic temporal lobe abscess was encountered. It may well be that since the use of penicillin they are either less frequent or their early treatment prevents them from developing into the severe acute forms, or the chronic encapsulated ones that reach the neurosurgeon.

Let me now discuss in more detail the method we advocate for dealing with brain abscess, first in acute, and then in chronic cases.

1. ACUTE ABSCESS

To the neurosurgeon, an acute abscess is not merely an abscess seen in the early days of its development. It is more than that. It is an abscess with high intracranial pressure, threatening life during the first 2 to 3 weeks of its course. In other words, an acute abscess behaves like a tumor with herniation of the temporal lobe through the tentorium.[5] Death results more from pressure on the brain stem than from infection. But it must be emphasized that, while the temporal lobe herniation is due in part to the abscess itself, it is due mainly to oedema of the white matter; and in this oedema, infection plays a very important part. In the average case, oedema surrounding an

acute brain abscess is much more pronounced than oedema surrounding an acute brain tumor. In some cases an acute meningitis is present when the acute brain abscess is developing.

Anatomically an acute abscess is not yet fully encapsulated. As a rule, we think now that 3 weeks is the average time for a well defined capsule to form. Often enough it was found that only 2 weeks had elapsed between the first clinical sign of abscess and the formation of a thin capsule. In a typical acute abscess, the capsule is present only in some places or not at all. The central part consists of pus, the amount of which may vary from a few drops to 15 cc. The white matter is very oedematous, as noted above.

Acute brain abscesses are young abscesses, but many young abscesses will not exhibit an acute stage, at least clinically. They develop without intracranial pressure until they reach the chronic stage.

When confronted with a drowsy patient a few days after the onset of headaches, fits, or mental disturbances during the course of an infectious process, a brain abscess is to be feared. A burr hole should be made in the skull over the most probable site. In some cases several burr holes may be required before tapping the abscess; but in any case it is a much safer procedure than a ventriculogram, where there is risk of increasing cerebral oedema and brain-stem pressure. A large blunt needle is used to feel more readily if there is or is not a capsule. When the abscess is located it is tapped and the pus removed. It will often come more freely by aspiration. Penicillin (10,000 or 20,000 units) is instilled into the abscess cavity, and the organisms are identified. Subsequently three situations may be anticipated.

(1) The abscess yields a fair quantity of pus (e.g., 10 cc.). Usually, with penicillin, the patient will improve enough to be able to wait for the formation of a capsule, sometimes after several aspirations. Later, the abscess, then chronic, is extirpated "en masse" when the patient is in better condition. As Kahn[4] pointed out, instillation of thorotrast into the abscess is very helpful in following the progress of encapsulation.

(2) Sometimes, usually after several aspirations and instillations of penicillin, the abscess seems to disappear, and a permanent cure may be brought about through this simple procedure. However, in our experience of "neurosurgical" acute cases, this is a rare occurrence. Such cases were described during the sulfa age and even before it, especially in children, but in any case, before assuming a complete cure, a ventriculogram should be performed. This will show usually a fairly large scar with shift of the ventricular system. Even without recurrence, epileptic fits are greatly to be feared in the future. On the other hand, the possibility of recurrence cannot be ruled out for some elements of capsule containing the organisms may already have been formed.

(3) Frequently the abscess yields only a few drops of pus. It is our impression that penicillin locally and intravenously will not be very effective. In any case, as a working rule we suggest waiting about 24 hours. If, after this time, signs of intracranial pressure have not improved, something more

must be done. Clovis Vincent's decompressive flap over the abscess is sometimes sufficient. Cairns, in one instance at least, went further and opened the dura. Now, with penicillin, we believe that it is possible and necessary, more often than not, to take a drastic course: turn a flap, open the dura over the abscess, and then take out the whole acutely inflamed zone along with a generous part of the surrounding oedematous white matter, opening the ventricle if it happens to be met. Then 20,000 units of penicillin are placed in the cerebral cavity and 20,000 in the ventricle; 100,000 units are given intramuscularly and intravenously for 4 days, and 20,000 under the flap for 4 days. We believe this procedure is the best to lower intracranial pressure and reduce temporal herniation. Of course better results may be expected with, for example, a temporal lobe abscess than a Rolandic where a less extensive extirpation is permissible and less reduction of temporal herniation results.

There were 4 cases of acute abscesses in our series of 17. The first, a left frontal abscess, was metastatic from a tonsillar infection. It was a corticomeningeal or perhaps as well described as a meningeal abscess, between dura and cortex. Gram-positive bacilli were isolated. The patient was comatose when first seen, and the abscess was tapped and aspirated. We had no penicillin at that time (one month after the liberation of Paris) and the patient died 2 days later with a diffuse meningitis.

The remaining patients with acute abscess, treated with penicillin, were all cured. The second patient had a temporal abscess, metastatic from a carbuncle of the neck. The classical method of Vincent was followed at first, but after 2 weeks, in spite of the decompressive flap, the patient again became comatose. At operation three temporal lobe abscesses were found, which were completely extirpated with approximately the anterior half of the temporal white matter. It should be noted that at least one of the abscesses had no capsule, and that the ventricle was opened. With penicillin locally and intravenously complete cure was obtained, but the flap had to be sacrificed due to staphylococcal osteitis.

The third patient had an acute right temporal abscess, developing at the same time as an acute diffuse streptococcal meningitis following measles. Both conditions were cured by penicillin introduced directly into ventricles and abscess. Later, this child developed chronic frontal abscesses (see below), and a permanent cure was achieved by complete extirpation. However, the acute temporal abscess had been completely cured by tapping and penicillin.

The fourth patient with acute abscess, completely cured by extirpation and penicillin, was that described above in Case 1, in which the abscess was metastatic from the lung. May I emphasize again that, in this patient, tapping and penicillin did not bring about any significant improvement.

2. CHRONIC ABSCESS

A chronic abscess is always surrounded by some sort of capsule, usually thick enough to be dissected out. The capsule is part of the disease and must

not be left in the brain, for, not only does it contain organisms but it behaves like a foreign body. That is why all drainage methods are definitely inadequate. Either suppuration never stops or, even with penicillin, it lasts for months, and one can never be sure of having achieved a complete cure. On the other hand, complete extirpation results in quick and permanent cure through the use of penicillin in every case.

Before the war it was thought that, on the average, 4 to 6 weeks are necessary for the formation of a definite capsule. Now it is our experience that 3 weeks is long enough in many instances. The chemotherapy that most patients now receive before reaching the neurosurgeon most likely accounts for this shorter period for encapsulation.

As a rule chronic abscesses exhibit less alarming signs than acute ones. Intracranial pressure is usually apparent before it has attained a very high level, and the patient is conscious when the abscess has to be dealt with. This does not mean that the outlook is not serious. On the contrary, many of these chronic abscesses were major problems; before the war, in our experience, about 40 per cent of the patients died.

Chronic abscesses may be divided into two very distinct groups, depending on the capsule itself: regular abscess and irregular abscess.

1. *Regular Abscess.* In our series of 13 chronic abscesses, only 3 regular ones were encountered. These are the classical encapsulated abscesses, usually smaller than a hen's egg, situated in the white matter, sometimes quite close to the ventricular wall. Their most important feature, however, is the capsule itself which is everywhere thick enough and smooth enough to be dissected out with little difficulty. Abscesses of this kind following an infection such as frontal sinusitis are usually attached to the dura by firm adhesions at one point. It is generally possible, however, to avoid opening the abscess. When they are too large, aspiration can reduce their size, and the opening in the capsule is readily closed. There are few new developments in the treatment of this class of regular abscess. This is the type upon which Clovis Vincent's method was based: extirpation "en masse" without opening the abscess and of course without drainage. Even before penicillin regular abscesses were cured in this way, although an osteitis of the flap was sometimes unavoidable, if the abscess had first been aspirated.

In all 3 of our cases of regular abscess encountered in the past 15 months treatment was by extirpation "en masse" and penicillin. The abscesses were not opened. No complications developed, and all patients were quickly and permanently cured.

The first case was a large abscess in the right prefrontal lobe (Fig. 4), following an ethmoidal fracture. Organism: Streptococcus.

The second was a right frontal streptococcal medium-sized abscess following a diffuse meningitis (Figs. 5 and 6). This same child had first an acute temporal abscess described above. The right frontal chronic abscess was removed "en masse" and cured without incident. This same patient also de-

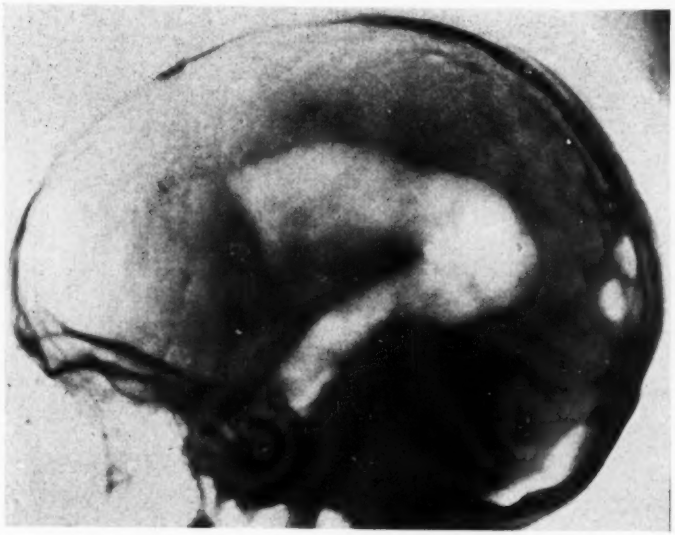

FIG. 5. Case of chronic right frontal regular abscess following meningitis. The patient was a child who previously had had an acute temporal abscess and subsequently had four left frontal abscesses (see Fig. 6).

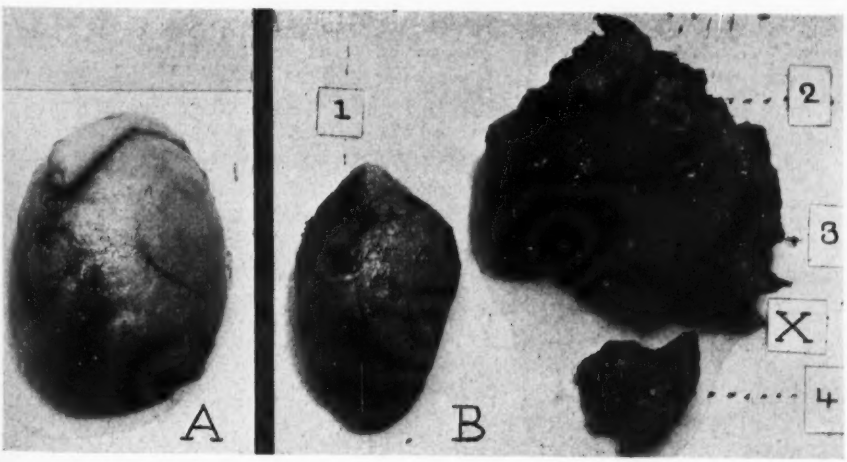

FIG. 6. Abscesses in patient with meningitis (see Fig. 5). (A) right frontal regular abscess, followed by (B) four left frontal abscesses: (1) regular; (2, 3 and 4) irregular. (X) Fibrous formation.

veloped left frontal irregular abscesses which required treatment and are described below.

The third case was a large left frontal abscess following a tangential frontal bullet wound (Fig. 7). Organisms: Staphylococci and gram-positive bacilli.

In those cases where the ventricle has been opened, even though the abscess has not, penicillin should be placed in the ventricle as well as into the cerebral bed of the abscess.

2. *Irregular Abscess.* These were by far the more common in our series, comprising 10 out of the 13 chronic cases. The abscesses may be single or multiple, and in the latter event are closely connected to each other. The most important feature is irregularity of the capsule. First, there is irregularity of shape: far from being a regular spherical or ovoid mass, it is asymmetric, limited by surfaces here concave and there convex. Then there is irregularity in thickness: in some places a "normal" capsule is met, about ½ cm. in thickness; in other places it may be a very tough wall 2 to 3 cm. thick; and in still other areas the capsule may be so thin that it is practically non-existent. This last and very important aspect is frequently found in the deepest parts of the abscess, especially close to the ventricular wall, where there is often no real limiting wall between pus and ventricle except the ependyma. These findings are in accord with the experimental work of Falconer, McFarlan and Dorothy Russell.[3] Irregularity of capsule in this series could not be shown to have any direct relationship to special organisms or to the age of the abscess, although it may be said that the older the abscess, the thicker (in places, but not everywhere) the capsule will be. The size of an irregular abscess or group of abscesses is usually, though not always, large, and on the average larger than a hen's egg. As a result, at least at one pole these abscesses are likely to be in close proximity to the basal ganglia and brain stem. As the thickness of the walls is so variable, the external size of the abscess is no indication as to the amount of pus it may contain. Finally, it is often very difficult to define the limits of such irregular abscesses: the outer surface of the capsule is frequently closely connected with fibrous masses which may or may not be new abscesses in formation, but which, in any case, it is our policy to remove. Some of these are illustrated in Figs. 1, 2, 3, 6, 8, and 11.

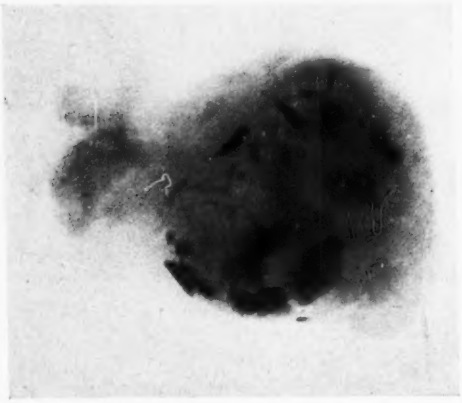

FIG. 7. X-ray of chronic left frontal regular abscess showing bone fragments. The patient had had a tangential frontal bullet wound.

These anatomical features of irregular abscess lead to important surgical considerations. As a rule tapping and aspiration do not reduce their volume significantly. In our opinion it is perfectly useless to try any extirpation "en masse" without opening the abscess. Firstly it is impossible because the capsule does not exist in some places, usually at the more dangerous areas near the ventricle. Secondly it is not always desirable. Indeed, these large irregular abscesses with all the surrounding fibrous adhesions, often in close contact with important brain structures, are not unlike large deeply situated meningiomas. In other words one must attempt to remove them without any untoward damage to brain function. This can only be achieved through deliberate opening and even fragmentation of the abscess. Excision of the abscess, its capsule, contents and fibrous adhesions must be thorough and complete to rule out any possibility of recurrence and to leave as neat and simple a scar as possible to minimize the possibility of postoperative epileptic fits. In irregular abscesses this complete extirpation should be done, one might say, as well from inside as from outside. If the ventricle has to be opened, which is usual, it should be opened not accidentally, but deliberately, with all suitable precautions.

Before penicillin, this drastic method of dealing with irregular abscesses was practically impossible without diffuse postoperative meningitis. With penicillin, we felt it should be tried and, as will be seen, this has been carried out successfully in 8 out of 8 cases. But before giving particulars of the case reports, it must be pointed out that the distinction between regular and irregular abscess is essentially a practical one. A very large regular abscess should be treated as an irregular one, if aspiration does not yield much pus. On the other hand, irregular abscesses, when small, need not be taken out piecemeal, though they might be open in some places. The whole problem depends on such factors as size, shape, site, and toughness of capsule and can be solved only during the operation. The rule to be followed is: whatever a chronic abscess is like, it must be taken out completely, even if this means one or several openings in the abscess.

Penicillin is given as follows: 200,000 units daily intravenously and intramuscularly for 4 days; 20,000 units into the abscess bed, and the same dose into the ventricle at operation; 20,000 units under the flap for 4 days. Sometimes, if gram-negative bacilli are found we have of course used sulfadiazine (we have not as yet tried streptomycin).

The first 2 patients in our 10 cases of irregular abscesses were operated upon before we had penicillin and both died. One had 2 huge traumatic right frontal abscesses (streptococci and staphylococci), and died 12 days later without meningitis. We feel that complete extirpation was carried out without enough fragmentation, and death occurred as in some cases after excision of very large tumors close to the third ventricle. The other patient had a group of gangrenous left parietal traumatic abscesses (staphylococci and B. perfringens): death, a week later, was due to meningitis.

In the following 8 cases the patients were completely cured and, on an average, left the hospital in a month's time.

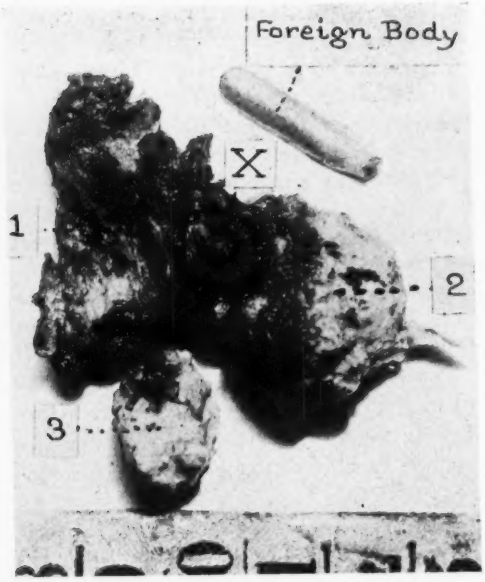

FIG. 8. Three chronic left parietal irregular abscesses, which followed a craniocerebral wound with retained foreign body. (X) Fibrous formation.

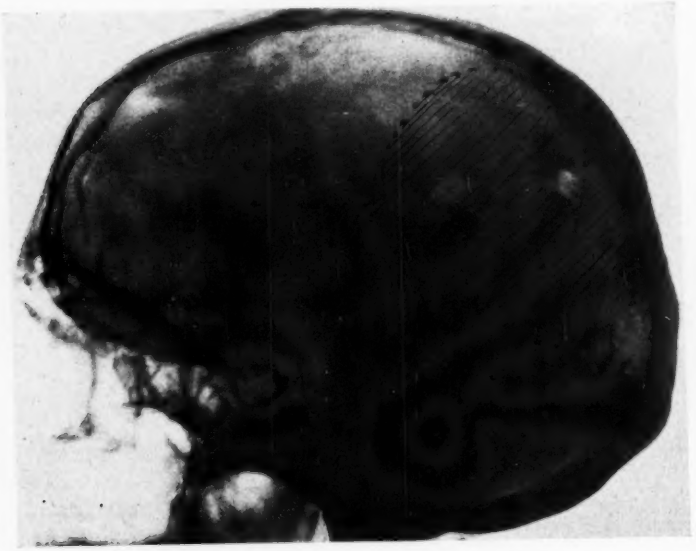

FIG. 9. Roentgenogram showing site of huge chronic right occipitoparietotemporal abscesses of irregular type extirpated Oct. 13, 1945. The patient had had a bullet removed from the corpus callosum Dec. 1, 1944. (See Fig. 2 for photograph of the abscess.)

FIG. 10. Case of two chronic cerebellar abscesses following mastoiditis (see Fig. 11). Abscess is present in upper half of right cerebellar hemisphere.

1. Three small abscesses, following a right temporal wound. Ventricle opened. Colon bacilli. Sulfamidotherapy locally and orally.

2. Three left parietal abscesses following a craniocerebral wound with retained foreign body (Fig. 8). Staphylococci. Osteitis of flap. Tantalum plate inserted later.

3. Four left frontal abscesses developing as a complication of streptococcal meningitis in the same patient who had first an acute right temporal, and then a chronic regular right frontal abscess (Fig. 6). Child has been well since (7 months).

4. This is the metastatic abscess from the lung described in Case 2.

5. A huge group of abscesses, as large as one's fist, following removal of a bullet lying on the corpus callosum (Figs 2 and 9).

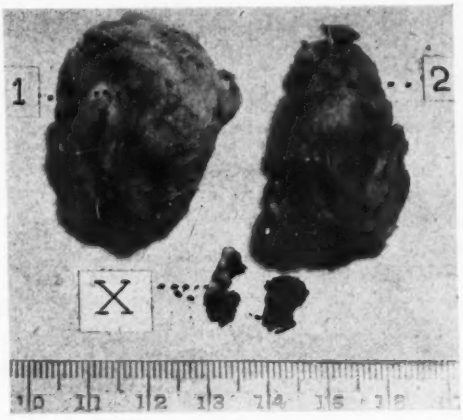

FIG. 11. Showing the two chronic cerebellar irregular abscesses (see Fig. 10). (X) Fibrous formation.

Abscesses extended from right occipitotemporal cortex to falx and corpus callosum. Capsule in places was several cm. thick (8 months history). Staphylococci. Ventricle opened. Well since (6 months).

6. Two right cerebellar abscesses following mastoiditis (Figs. 10 and 11). One abscess was open and in contact with cerebrospinal fluid of the lateral cistern. Streptococci. Patient left hospital 3 weeks later without the slightest sign of postoperative infection.

7. This was a rather peculiar case, which could be classified as either a meningeal, or a cortical abscess. It was a left Rolandic group of abscesses following infection of the left eyelid. Streptococci and colon bacilli. Sulfadiazine was used in conjunction with penicillin. Patient is now well, but it is too early to be certain of complete cure.

8. This is the last metastatic abscess from the lung, described in Case 3.

SUMMARY

We advocate the principle of complete extirpation of brain abscess in one stage, whether acute or chronic. With penicillin, this is possible in most cases without the development of meningitis, even though it may require opening the abscess and often the ventricle. If organisms not sensitive to penicillin are encountered, other drugs should be used.

The report comprises 17 cases of brain abscess. In the first 3 cases, before penicillin was available, the patients did not survive. In the remaining 14 cases, in 13 of which penicillin was used, the patients have all been cured in one stage. None of the abscesses was drained. Of these 14, 10 were very serious forms, most of which would not have been cured otherwise. They include 3 thoracogenic abscesses which are described in detail.

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GUIDES FOR NERVE REGENERATION ACROSS GAPS*

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THE PROBLEM of how to bridge nerve gaps that cannot be further reduced by manipulative procedures (mobilization, rerouting, stretching, etc.) has not yet been adequately solved. The results of nerve grafts in man have been disappointing,[3,4,5] and though improvements in technique or the use of devitalized undenatured grafts [10,15] might yet bring better success, the prospects are so uncertain that it is indicated to explore other alternatives. Several attempts in that direction have been made in the past. Various tissues, living or fixed, as well as artificial structures, organic or inorganic, have been tried as guides for regenerating nerve fibers, mostly without significant success. As has been pointed out in a recent review of this subject by Weiss,[12] most of the former failures can be explained by errors of technique, often based on misconceptions about the mechanism of nerve regeneration. We have, therefore, continued the search for improved methods on an experimental scale, and the results are summarized in this article.

On a previous occasion,[12] brief mention was made of experiments on the bridging of nerve gaps by arterial tubes filled with blood. While resembling superficially some older methods of "tubulation," these experiments observed more closely the known requisites of nerve regeneration, and hence, yielded markedly better success. A more detailed account of these experiments is presented in the following.

TUBULATED BLOOD BRIDGES IN THE RAT

Pieces were cut out from various nerves of rats (sciatic, peroneal, vagus) leaving gaps of about 10 mm. in length. The nerve ends were then fitted into a tube of artery of proper length and width by the technique described previously.[8] The empty part of the tube was then filled with blood or some other medium to be tested, guarding against the introduction of air bubbles. Experience has shown that fillings of Ringer's solution or blood plasma lead to fibrosis with obstruction of nerve regeneration. Therefore, only gaps filled with whole blood will be considered here.

Seven rats thus operated upon were biopsied after from 4 to 17 weeks. The experimental nerves were studied histologically (Bodian's silver impregnation). In the older cases, recovery of function had occurred but was decidedly inferior to that observed after end-to-end union or even after the use of frozen-dried grafts.[15] Aside from technical accidents (slipping of one of the nerve ends from the tube in one case), the method had inherent defects, which were revealed by the microscopic study.

* This work was done under the direction of Dr. Paul Weiss under contracts between the Offices of Scientific Research and Development (Committee on Medical Research) and of the Surgeon General, United States Army, and the University of Chicago. It was also aided by the Dr. Wallace C. and Clara A. Abbott Memorial Fund of the University of Chicago.

In a number of cases, the blood-filled part of the artery had collapsed, presumably because of leakage; the wall was creased longitudinally, with practically no lumen left. A narrow strand of dense connective tissue, sometimes containing a large longitudinal blood vessel, occupied the limited central space, and regenerating nerve fibers had not been able to penetrate it.

In cases in which the lumen had remained wide open, nerve regeneration across the gap had proceeded satisfactorily for varying distances, but then been impeded by islands of heavily fibrosed or areolar tissue. As already noted in previous similar experiments,[17] the orientation of the cellular strands and nerve fiber bundles was remarkably straight.

On the basis of earlier experiments,[16] we thought that the formation of fibrous blocks could perhaps be prevented by increasing the fibrinolytic potency of the initial clot through the admixture of large masses of leukocytes, taken from the buffy coat (stratum between red cells and plasma) of centrifuged homologous blood.

TUBULATED BLOOD BRIDGES IN THE CAT

In five cats (F7–11), pieces were cut out from the tibial or peroneal nerves in midthigh, leaving the stumps after retraction separated by gaps of 20, 18, 10, 28 and 25 mm., i.e., fifteen to thirty times as long as the width of the nerves. The ends were introduced into a matching segment of carotid artery, which was then filled with the modified blood. Except for cat F11, in which one nerve end was found at autopsy to have slipped out, the operations and subsequent recovery histories were satisfactory. Brief case descriptions follow.

Cat F7. Gap in tibial nerve, 20 mm. long, bridged with carotid artery (kept refrigerated in Ringer's solution for 3 days), filled with blood plasma and erythrocytes from centrifuged heparinized blood in a volume ratio of 2:1 and an added amount of buffy coat.

At 58 days p.op., the gastrocnemius was well palpable, but active function was doubtful. Active plantar flexion was definitely present at 105 days p.op.; it was still subnormal in strength at 202 days, but well recovered after one year; even then, the animal, digitigrade for most of the time, would occasionally relapse into a few plantigrade steps.

At biopsy (393 days p.op.), the artery was found to have narrowed so as to cause a "bottleneck" with consequent bulbous enlargement of the proximal stump;[9] there was no distal swelling. The muscle weights were 27.8 gm. for the tibial group, and 8.3 gm. for the peroneal group. Since the opposite leg had been subjected to another operation, it could not be used as control for muscle weights. But the weight ratio of 3.35 between tibial (reinnervated) and peroneal (undisturbed) muscles in the experimental leg is approximately the same as that of normal legs, hence indicates full weight recovery of the reinnervated muscles.

Microscopic examination: The nerve in the area of the former gap has a characteristic pattern, differing from that observed after regeneration over short gaps,[8] but even more strikingly from that noted in gaps filled with scar tissue. It differs from the former in that it is less well oriented and contains more connective tissue, but its constitution is much nearer that of normal nerve than would ever be obtained in ordinary scar tissue. The regenerated nerve fibers are grouped into large bundles which are separated by partitions of connective tissue thicker than regular perineuria. The general direction of these bundles is longitudinal, but not straight; they interweave and give the nerve a braided appearance. Fig. 1 shows a longitudinal section through this region. The most significant difference between this type of regeneration and that in ordinary scars is that the nerve fibers are assembled into well

organized strands with plenty of rather liquid (light colored in Fig. 1) spaces for the expansion and addition of new fibers, and that the connective tissue runs parallel to, and in between, the channels, instead of forming solid blocks across. The transition between this new bridge tissue and the old nerve stumps is continuous and smooth. Only at the transition to the distal stump, the tissue assumes a denser consistency, with collagen deposits inside the nerve bundles. The distal stump itself was well filled with regenerated fibers, and sample cross sections (osmic acid) at various levels down to the ankle reveal abundant reinnervation and good myelinization. Fiber sizes in these distal nerve samples, however, were subnormal, with the majority of fibers measuring less than 3μ, a fair number up to 5μ, and only a few between 5 and 7μ. This deficit is evidently a result of the compression to which the fibers are subjected

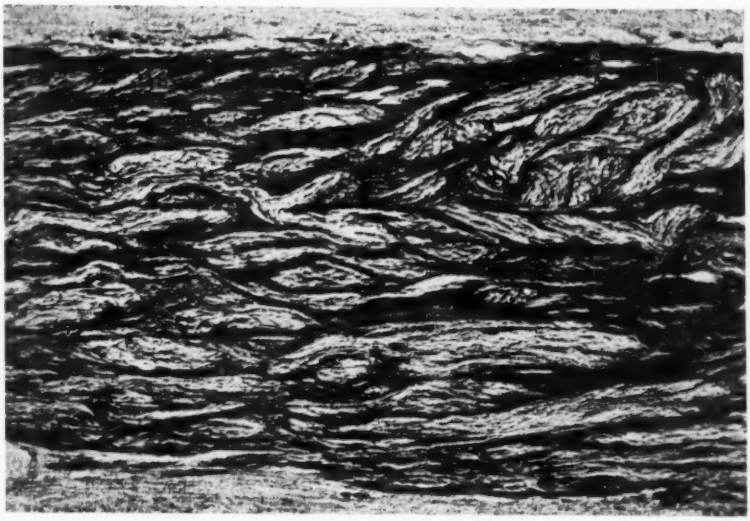

FIG. 1. Longitudinal section through the whole width of the regenerated nerve portion in the region of the former gap (F7), showing braiding of nerve bundles. Bodian and Mallory stain, ×98.

in the more compact fibrotic zone just mentioned, since lack of space for expansion anywhere along the course of a nerve fiber prevents the whole farther distal portion from growing in width.[18]

Cat F8. Gap in tibial nerve, 18 mm. long, bridged with kitten aorta filled with local blood. It was attempted to add some buffy layer to the clotting blood through a side branch of the aorta, but upon withdrawing of the pipette most of it spilled back, so that the gap filling in this case consisted essentially of unmodified blood. At 8 weeks p.op., signs of recovery appeared as feeble plantar extensions and palpable gastrocnemius contractions. Five weeks later, the animal died.

Autopsy: The nerve lay in a very smooth bed with no major adhesions. Upon slitting the sleeve longitudinally, a smooth cylindrical nerve segment was found to have formed over the former gap. No demarkation could be seen between this new segment and the former stumps. The weight of the calf muscles was still only 40 per cent of that of the control side, three months being too short a time for complete recovery.

Histological examination: Nerve fiber regeneration of normal density, oriented essentially longitudinally, in bundles braided as described in the preceding case. Some endoneurial

fibrosis, which had affected the diameter, but not the number, of the regenerated fibers. The distal stump was well neurotized.

Cat F9. Gap in peroneal nerve, 10 mm. long, bridged with coeliac artery of kitten, filled with whole blood and buffy coat from heparinized kitten blood; sleeve sealed to epineurium of stumps by diathermic electrocoagulation. Eight weeks p.op., toe spreading and withdrawal dorsiflexion had reappeared and kept steadily improving in strength. Biopsy after one year showed moderate adhesions and slight bulbous enlargement of the nerve indicative of a too tight sleeve. Muscle weight was fully recovered (weights of the combined peroneus and tibialis anticus muscles: 11.4 gm. on the experimental, 11.3 on the control side).

Histological examination: Full volume of fiber regeneration with little weaving and fairly straight longitudinal orientation (Fig. 2). Only a trace of fibrosis near entrance to distal stump; the latter fully neurotized. The superiority of this case over the two previous ones in

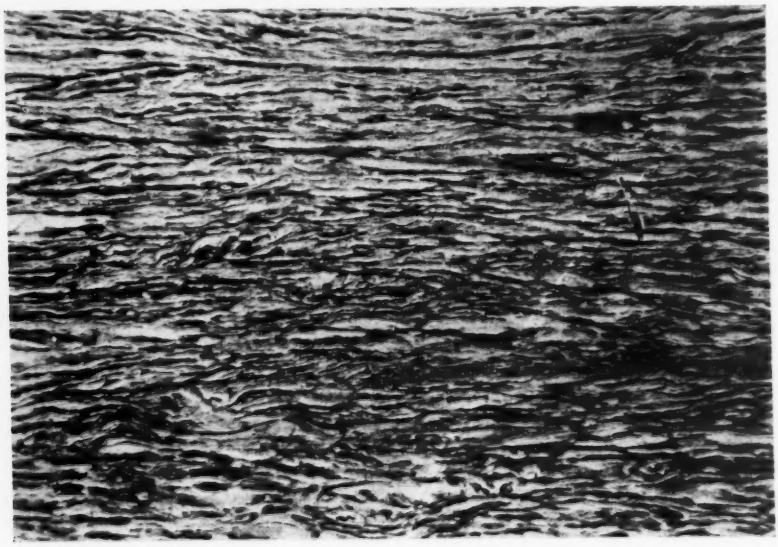

FIG. 2. Portion of longitudinal section of regenerated nerve segment in the former gap region (F9), showing straight fiber course. Silver impregnation after Bodian, ×185.

regard to straightness of the fiber course can be correlated with the shortness of the original gap (10 mm.).

Cat F10. Gap in peroneal nerve, 28 mm. long, bridged with kitten carotid filled with a mixture of plasma and erythrocytes from heparinized centrifuged kitten blood in a ratio of 1:4, with additions of buffy layer. A tantalum wire loop, threaded through the nerve stumps for holding purposes, was removed 8 days p.op. At that time, the artery appeared somewhat flattened. At 10 weeks p.op., dorsiflexion was definite, toe spreading indicated. At 19 weeks p.op., when the animal was sacrificed, function was recovered, though still somewhat weaker than on the control side.

Autopsy: Distal two thirds of the sleeve appear partly collapsed. Weight of dorsiflexor muscles (peroneus and tibialis anticus: 5.65 on experimental side, 7.95 on control side, i.e., only 71 per cent weight recovery.

Histological examination: Longitudinal folds of sleeve are evidence of the partial collapse

mentioned above; however, unlike the more severe cases of collapsed sleeves described in earlier reports,[8,14] the lumen had remained sufficiently wide to accommodate copious nerve regeneration. The fibers are again grouped into small fascicles separated by fibrous partitions and slightly braided. But, on the whole, the fiber course is remarkably straight and unconfused (Fig. 3). No fibrotic foci have developed in this case. Cross sections through the regenerated nerve at the ankle (osmic acid) show most fibers still of relatively small diameter, few above 3μ. (Regeneration distance from cut to ankle was 160 mm., total regeneration time 131 days.)

FIG. 3. Portion of longitudinal section of regenerated nerve in former gap (F10), showing straight fiber course without much braiding. The wall of the arterial sleeve can be seen. Bodian, ×190.

These results prove that gaps of several centimeters can be successfully bridged if the nerve fibers are offered an oriented bed of blood with augmented liquefying potency, contained in a straight tube. The regenerated nerve contains large streams of fibers, not merely those insignificant trickles occasionally found in the scars between separated, but unbridged, stumps. Since there has been no appreciable decline in the volume of regenerated fibers over gaps of nearly 3 cm., there is no reason to doubt that regeneration would have continued over even greater distances. However, the technical difficulties, particularly the danger of collapse, would mount rapidly with increasing length of the free portion of the sleeve. It was mainly to meet these difficulties that we began to modify the method in the following manner.

TUBULATED BLOOD BRIDGES WITH FIBER CORES

Experiments carried out with peripheral nerve fragments in tissue culture have shown that Schwann cells tend to adhere to the surfaces of artificial fibers of glass or synthetic resins even when the latter are embedded in

a clot of blood plasma.[13] The cells move out rapidly in tandem strands strictly aligned with the fiber axis. Since regenerating axons, in turn, tend to advance along Schwann cell strands, extension of these tissue culture experiments to problems of actual nerve repair suggested itself. We therefore supplemented the simple tubulation technique described above by including in the blood bridge large numbers of thin, parallel, longitudinally oriented artificial fibers of various kinds, for the double purpose of giving the link greater consistency and of giving the Schwann cells and axons oriented pathways leading straight from stump to stump.

EXPERIMENTS IN RATS

Most of the fibers used measured less than 0.03 mm. in diameter. They were of the same types as those used in the earlier tissue culture tests,[13] namely, glass wool (8μ or 26μ), Nylon, textile rayon, cellulose acetate, and "Cordura" (tire rayon).* These fibers were combed into parallel strands, cut to proper length, and inserted into the lumen of a somewhat longer sleeve of artery mounted on a special stand and filled with Ringer's solution. The fibers were dispersed by gentle manipulation, and the Ringer's solution was then gradually replaced by whole blood. After this had firmly clotted, the whole preparation was used much as if it were a nerve graft. A segment was excised from the peroneal nerve so as to leave a gap of *ca.* 10 mm., and the two stumps were inserted in the usual manner into the open ends of the arterial sleeve until they met the fiber-blood core. No further means of attachment were used.

The 24 animals included in this series were sacrificed between 7 and 77 days p.op. Our main purpose was to study the microscopic picture; all bridges except those containing glass fibers could be sectioned, as the fibers either dissolved or softened during the histological treatment. No motor recovery had occurred, and stimulation of the distal stumps at autopsy gave no evidence of sensory fiber regeneration. Functionally, these cases were complete failures. Microscopic examination revealed the reason, which was a purely technical one.

The artificial fiber core had failed to form an intimate union with at least one, and often both, stumps. It was separated from them by a small, but distinct, cavity filled with liquid. As neither spindle cells nor nerve fibers cross liquid gaps, no organic junction had been effected. The explanation of this non-union lies in the relative rigidity of the fiber core. As the blood menstruum in the sleeve became dissolved, the fibers were forced closer together into a rather stiff bundle, which then acted as a mechanical unit. While the arterial sleeve was elastic, the fiber core was not. The movements of the animal, therefore, caused continual slight displacements between the fiber bundle and the nerve stumps, and any incipient tissue links were thus constantly ruptured before they had a chance to become consolidated. The inset could thus become firmly attached to one stump or the other, but not to both.

In those cases in which the graft had become firmly fused with the proxi-

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* The fiber samples were obtained through the courtesy of Dr. M. M. Brubaker of the Experimental Station of E. I. duPont de Nemours and Company.

mal stump, it was invaded by regenerating nerve fibers in various ways and degrees. When the filaments of the core were too tightly packed, no tissue penetrated between them. When spaced more loosely, the interstices became invaded by a tissue composed of blood cells, mesenchyme, Schwann cells, nerve fibers and serous spaces, in varying ratios. Textile rayon evoked in all cases a strong foreign body reaction with small-cell infiltration, liquid exudates, and poor growth of organized tissue. Nylon, cellulose acetate and "Cordura," on the other hand, were much better tolerated. There was little or no liquefaction, and the regenerating tissue adhered firmly to the fiber surfaces. The cells and axons followed strictly longitudinal courses, not only where they were in direct contact with the guide fibers, but also in the intervening meshes. In some cases, the interior of the fiber bundle had remained devoid of cells, while there was ample growth in the more superficial layers, and these, in turn, were surrounded by a layer of pure regenerated nerve tissue, which filled the space between the fiber core and the wall of the sleeve. Similar observations were made in the cat experiments and will be reported below.

Even apart from the technical defects, nerve regeneration in this series cannot be rated as superior to that observed in rats with tubulated blood gaps without fiber cores.

EXPERIMENTS IN CATS

A. *Unattached fiber cores*

This series followed the same general plan as the previous one in rats, except that only glass fibers were used as cores, and collagen tubes, instead of arteries, for sleeves. These tubes, spun from fibers of dissolved and reprecipitated bovine collagen by a process developed at the Massachusetts Institute of Technology under the direction of Dr. F. O. Schmitt, were prepared for our nerve repair work according to our specifications. Both tanned and untanned tubes were available. The former are relatively unelastic, produce some slight tissue reaction, but persist for long periods without change, while the latter are highly elastic, but rather rapidly resorbed. We have made comparative studies on the relative merits of the various collagen sleeves in ordinary nerve splicing experiments, but have been unable to reach final conclusions because of the great number of variables involved (variability of the preparations, animals, operative incidents, etc.). For the present experiments, we used plain untanned collagen because of its greater elasticity.

In six of the seven animals of this series, the sleeve was filled with whole blood, in the seventh with gelatin. Gaps in the peroneal or tibial nerves of from 10 to 20 mm. were bridged in this manner; the animals were biopsied between 11 and 31 weeks p.op.

Five of these cases were complete failures, again for technical reasons. As in the corresponding rat series, the rigid compacted glass fiber bundle had become attached to one of the stumps only. Moreover, the collagen

tubes underwent resorption at a time when their content would still have needed protection against loss of liquid and ingrowth of fibrous tissue.

In two other cases, a tenuous union had been established between the insert and both stumps, and this tissue bridge then acted as a foundation for the gradual building up by further regeneration along its sides of a good-sized nerve. In one of these cases (gap of 12 mm. in tibial nerve), plantar extension had returned after 15 weeks, and at biopsy (16 weeks p.op.), electric stimulation of the proximal tibial nerve produced vigorous contraction of the plantar flexors; the weight of the latter was only 15 per cent below that of the controls from the opposite side. The proximal two thirds of the glass core were intimately interwoven with the substance of the nerve, but the distal third came to the surface and protruded at an acute angle. In the other case, in which a gap of 17 mm. in the peroneal nerve had been bridged, toe spreading and dorsiflexion had reappeared 12 weeks p.op. Biopsy revealed a well organized cylindrical nerve segment that had formed across the gap and from which the glass core bulged laterally. Electric stimulation of the proximal peroneal produced toe spreading and dorsiflexion of medium strength, and sensory responses could be evoked by stimulation of the nerve 125 mm. distal to the original lesion. Histological examination of the distal stump in the lower leg showed abundant neurotization.

These two cases prove that the method as such may lead to good results, provided the bridge has effected a firm primary union with both stumps. Once this has happened and the outgrowing tissue has formed a straight oriented connection between the stumps, the glass bridge can gradually be dispensed with. Its protrusion from the repaired nerve is apparently due to secondary elimination. It remains undecided, however, whether the addition of a fiber core in these cases was of any real advantage; a direct comparison with the coreless tubulated blood bridge described above is not possible, because in the present series unmodified whole blood was used, which is not an optimal medium.

B. *Attached fiber cores*

The simplest way to avoid the hazards encountered in the preceding series and to secure a firm union between stumps and bridge is to sew the fiber strands directly through the stumps. This has the double advantage of bringing the regenerating tissue into intimate contact with the guide fibers and of linking stumps and fiber bridge into a continuous chain, but has the disadvantage of disrupting the architecture of the nerve stumps at the suture points. It developed that the advantages overbalance the disadvantages. Of several procedures tried, the following proved most satisfactory (Fig. 4).

Synthetic resin fibers are combed out and a few hundred of them are threaded as a single strand through a wide-eyed straight needle. The needle is thrust through the side of the proximal stump (P), several mm. above the gap, and then guided along the axis of the nerve until it emerges in the center of the proximal cut, as shown in Fig. 4*A*. A collagen sleeve (S) of proper dimensions is slipped over the distal stump (D) and gathered so as to leave the nerve

end free. After connecting the two nerve stumps by a single suture thread (E; Fig. 4*B*), fastened to the edges of the respective epineuria under some tension, the needle with the fiber cable is threaded through the center of the distal stump for about 1 cm. and finally brought out laterally, as shown in Fig. 4*B;* the fiber cable is then cut at both ends flush with the wall of the nerve. The holding suture (E) is then removed, allowing the nerve ends to retract slightly. This makes the ends of the cable slip into the center of the nerve and also tends to straighten the fascicles of the distal stump which have been pushed back by the friction of the cable when it was first drawn through. Next, a semicylindrical trough (G), fashioned from annealed tantalum foil[11] to fit the dimensions of the nerve, is placed under the gap, as shown in Fig. 4*C*. The trough is filled with Ringer's solution, in which the fibers, which had been clinging together, separate again. The Ringer's solution is then gradually replaced with blood

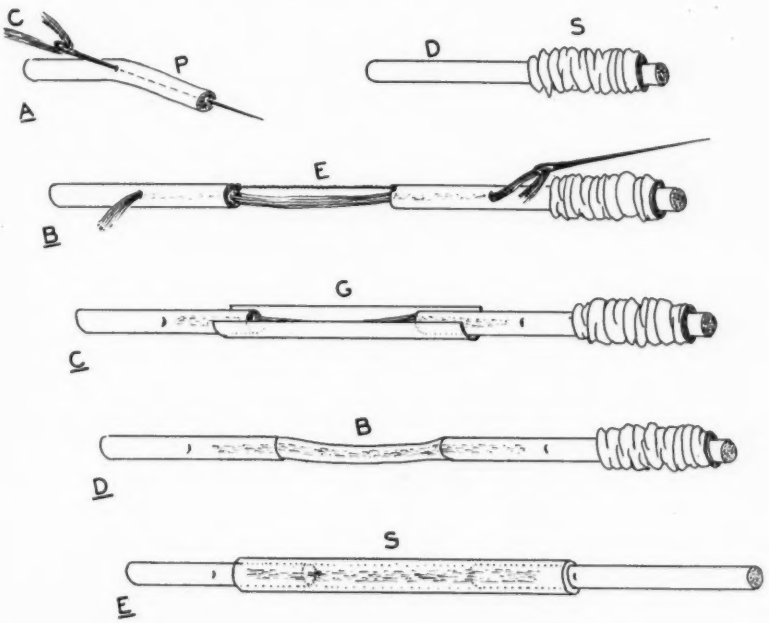
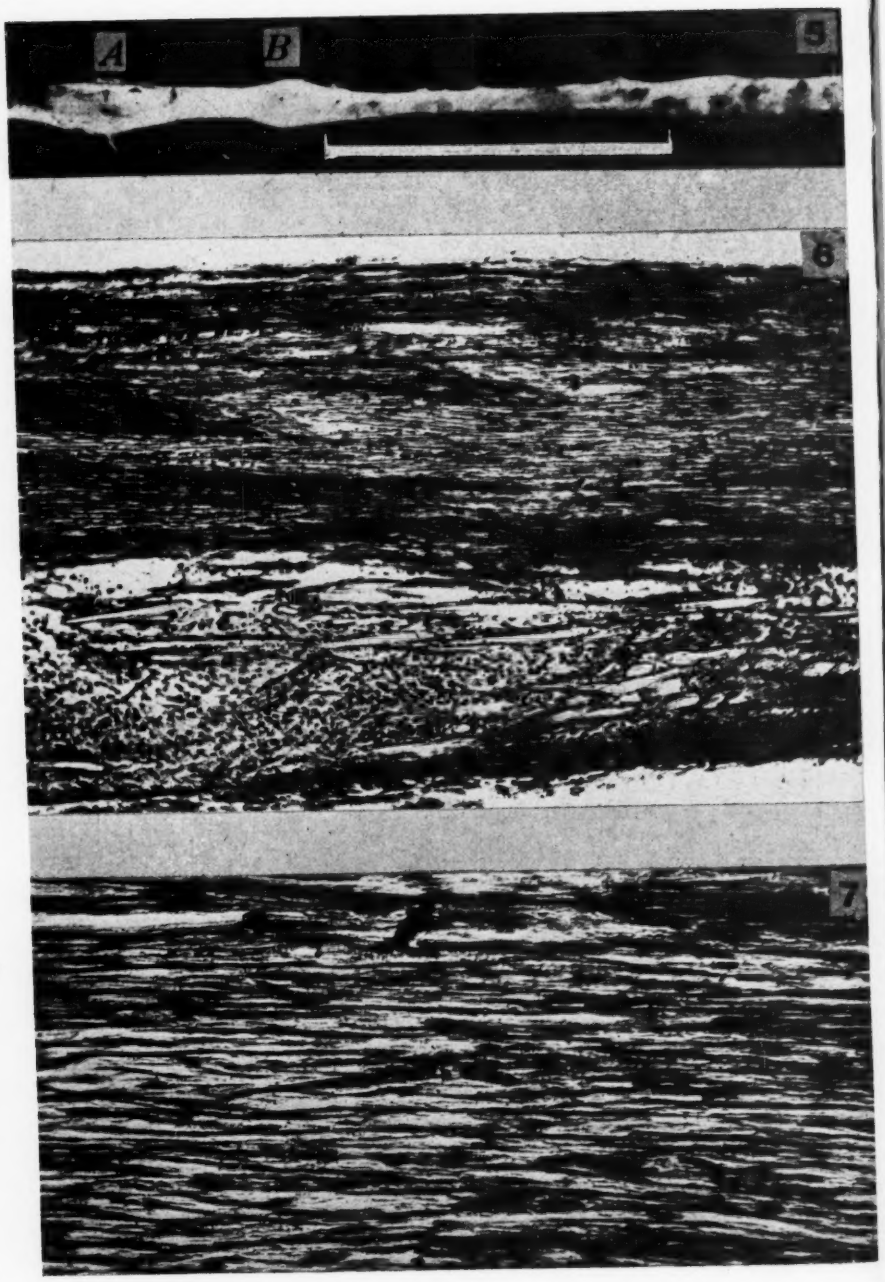


FIG. 4. Diagram of operation. Explanation in text.

taken from one of the local veins and dripped into the trough from a pipette. Excess blood flows off, but a clot soon begins to form along the fibers. After the whole content of the trough has firmly clotted, the trough is removed, leaving a blood segment (B; Fig. 4*D*) intercalated between the stumps. The collagen sleeve (S) is then pulled over the region (Fig. 4*E*). A description of three cases follows.

Cat F41. Peroneal gap of 21 mm. bridged with "Cordura" fibers (12μ diameter) in tubulated blood clot. Sacrificed after 20 weeks. Toe spreading and dorsiflexion were very well restored. Biopsy revealed a smooth cylindrical nerve (Fig. 5) with two slight bulbous swellings in the proximal stump, one (A) at the level where the cable had been drawn across the fascicles, and the other (B) just proximal to the entrance of the collagen tube, which evidently had been fitted too tightly. Electrical stimulation proved that no nerve fibers had escaped,



FIGS. 5, 6 and 7. See opposite page for description.

and there were only minor adhesions. Sensory responses could be elicited from the distal stump at a distance of 180 mm. from the level of the lesion, which is as far as the nerve could be dissected free. The dorsiflexors had recovered to 84 per cent of the weight of their opposite controls.

Histological examination (Fig. 6): The "Cordura" fibers lay excentrically in the newly formed nerve segment. A moderate foreign body reaction with small-cell infiltration is noticeable. Some regenerated tissue, consisting of fibrocytes, Schwann cells and some axons was present between the fibers, but it was relatively small in amount. In contrast, nerve regeneration around this fiber bundle, and particularly to one side, was as abundant as after end-to-end suture. It differs from that in coreless blood clots by the straightness of the fiber course (Fig. 7) and the absence of braiding and of fibrotic partitions and islands.

Cat F42. Peroneal gap of 28 mm. bridged with "Cordura" cable in tubulated blood clot. Sacrificed 20 weeks p.op. Toe spreading and dorsiflexion had recovered to nearly full strength; the dorsiflexors had returned to 87 per cent of the weight of the controls. Biopsy revealed moderate adhesions between the sheath of the new nerve segment and its surroundings, but upon dissection of the sheath a perfectly smooth cylindrical regenerated nerve appeared in the interior.

Histological examination: The filaments had remained more widely spaced than in the preceding case. Here and there strands of dense fibrous tissue with strictly longitudinal orientation separated the large and equally well oriented fascicles of nerve fibers. There was no trace of transverse fibrosis.

Cat F43. Peroneal gap of 19 mm. bridged with cable of Nylon fibers in blood clot in sleeve of frozen and thawed artery. Sacrificed 30 weeks p.op., when no functional recovery was noticed. Biopsy revealed that the distal nerve stump had slipped from the sleeve, the free end of which had then collapsed. The whole length of the sleeve up to that point, however, was filled with regenerated nerve.

Histological examination: Excellent straight nerve fiber regeneration in full volume has occurred along and between the Nylon filaments. Most of the latter are coated by thin endothelioid cell sheaths, but otherwise there has been no cellular reaction. There is no evidence of fibrosis. The histological picture leaves no doubt that if the distal stump had not pulled out by accident functional recovery would have been as good as after gapless nerve reunion.

In all three cases with fiber cables, the architecture of the nerve stumps was considerably disorganized at the points of suture, resulting in the formation of small local neuromas. But since these regions are continuous with the much less disturbed end segments of the stumps, where the cable runs parallel to the nerve axis, the effect on regeneration is no more serious than in an ordinary lesion "in continuity."

COMMENT

The reported experiments demonstrate the feasibility of bridging nerve gaps twenty to thirty times as long as the diameter of the nerve by methods other than nerve grafts. All techniques here described as more or less successful have two features in common: (1) the use of a connecting bridge be-

FIG. 5. Regenerated nerve of F41 at biopsy after removal of sleeve. The length of the former gap is marked by a bracket. For further explanation, see text. (×2.5)

FIG. 6. Part of longitudinal section of regenerated nerve in zone of former gap (F 41). "Cordura" cable with small-cell infiltration below; bulk of regenerated nerve above; weak nerve bundle also at lower margin of "Cordura" cable. (×100)

FIG. 7. Detail from regenerated part of nerve of Figs. 5 and 6, showing parallel orientation of regenerated fibers. (Bodian, ×455)

tween the stumps to serve indirectly as a trellis for the regenerating nerve tissue, and (2) the enclosure of this bridge, for reasons of orientation, consistency and protection, in an elastic tube. Let us consider these two provisions separately.

(1) The medium between the stumps must be so chosen as (a) to facilitate physically the permeation and resettlement of the gap with true nerve elements (Schwann cells, axons, etc.) of the distribution and orientation of typical nerve, and (b) not to impede chemically the regeneration process, either through toxic substances or by destroying physical guide structures indispensable for regeneration. The physical prerequisites referred to in (a) include (α) the presence of oriented interfaces (guide structures) along which cells and axons can advance, and (β) provisions for an adequate amount of liquid in the spaces between the guide structures. This liquid fulfills several functions: it enhances the association of nerve fibers into bundles ("fasciculation");[7] it provides space needed for the expansion of the regenerating tissue, particularly the growth in width of the nerve fibers;[12] and there are indications that it may also have an antifibrotic effect, since in our experience, there is a direct relation between compactness of the early union tissue and later fibrosis.

The bridges of coagulated blood described above satisfy both conditions (α) and (β). The fibrin framework furnishes the guide structures, and enzymatic liquefaction of the clot provides for the liquid spaces.[16] However, the longitudinal orientation of the fibrin was evidently less strict, and the liquid less abundant, than was the case in our earlier experiments with shorter gaps. Hence, the "braiding" of the nerve fibers, and with the use of whole blood, the greater incidence of fibrotic islands. It was in order to remedy the latter condition that we attempted to increase the liquid content of the union tissue by adding more leukocytes as proteolytic agents to the original clot. The results indicate that this had the desired effect, but the number of cases is too small to be fully conclusive.

The introduction of fiber cables into the blood bridges was intended to insure a straighter course of regeneration across the gap and to give the bridge enough consistency to prevent the collapse of the sleeve. Both effects were attained. The numerous parallel filaments caused the regenerating tissue to assume a corresponding longitudinal orientation, with the result that nerve regeneration became straight instead of wavy. The detailed mechanism by which the parallel organization of the bridge tissue is effected remains to be determined. But the effect as such is clear. Quite unexpectedly, the fiber cables served another useful purpose by increasing the liquidity of the union tissue in the sense outlined before. Some types of these synthetic fibers gave rise to a mild foreign body reaction, and the proteolytic activity of the accumulating leukocytes then produced automatically the effect achieved in the earlier series by the addition of buffy coat.

(2) Enclosure of the blood bridges in elastic tubes is necessary for a variety of reasons, most of which have been analytically discussed on pre-

vious occasions[8,12,16] and need not be repeated here in detail. Briefly, the sleeve makes it possible for the enclosed segment to form an oriented integral link between the nerve stumps and yet to retain morphological independence from the surroundings. The effects of the sleeve can best be appreciated by considering what would happen if the sleeve were omitted. Lacking insulation from the surroundings, the bridge would adhere to its bed all along the exposed surface, and the resulting disorganization of the stress pattern[12] would establish pathways both for the intrusion of fibrous tissue and for the dissipation of Schwann cells (glioma) and axons (neuroma). Moreover, the liquid referred to in point (1) would seep off, leaving the tissue in a state of progressive condensation. It should be pointed out that while adhesions between the outside of the sleeve and the wound bed are a common occurrence, the new nerve segment itself retains a smooth surface and slides freely within the sleeve—a fact noted particularly in the earlier observations with tantalum sleeves[11,20] and confirmed again in the present experiments.

The fact that sleeves also cut the regenerating segment off from outside sources of vascularization, has proved to be immaterial for the regeneration process under the given conditions. Blood supply is furnished entirely by vessels and capillaries sprouting from the stumps *pari passu* with the rest of the tissue. This supply is adequate. In this regard, blood gaps offer an advantage over live nerve grafts, which cannot maintain themselves unless promptly revascularized. As Tarlov and Epstein[6] have demonstrated, and as we have confirmed by *in vitro* experiments,[19] vascularization from the ends has to be supplemented by vascularization from the surface in order to permit a graft above a certain length to survive; sheathing of such grafts, therefore, impairs their viability. It should be noted, however, that such surface vascularization is needed for the preservation of the old tissue of the graft, and not for the newly regenerating tissue of the nerve. Necrosis due to inadequate blood supply renders a graft useless and even harmful. However, neither frozen-dried grafts, nor the blood bridges of the present series need oxygen for their maintenance; they may, therefore, be left devoid of blood supply for prolonged periods without becoming "necrotic" blocks to regeneration. This explains why sheathing, though definitely contraindicated around live grafts, is wholly innocuous with the present technique.

The sleeve affects the regeneration process by a combination of interrelated factors, and to describe it as if it were a pipe line in a gross mechanical sense, is not just a simplified expression, but an outright misrepresentation. Consequently, the practice of lumping all techniques of nerve repair in which sleeves of one form or another are used into a common class "tubulation" is wholly arbitrary, and if used as a basis for judgement, scientifically unsound. Many methods described as "tubulation" or under similar names have nothing in common but the name. Therefore, the success of the "tubulation" method—if anyone should care to designate it as such—described in this article is in no way at variance with the patent failures of other "tubulation"

methods. The idea of bridging nerve gaps by tubes, following the naive pipeline concept, is old. Except for the experiments of Kirk and Lewis,[2] which came close to the ones reported above, the results were bad, largely because either the material or the filling or the application of the tube was wrong. (For a detailed evaluation of these earlier attempts, see Weiss.[12]) The idea of guide structures over a gap is likewise not new. Assaky[1] spanned gaps of several cm. in sciatic nerves (four dogs and three rabbits) by a few catgut threads, which he left lying exposed in the wound bed. He observed heavy scarring, but nerve fibers had regenerated across the gap into the distal stump; how far, is not evident from his histological description. Although he reports functional recovery and response to electrical stimuli applied above the lesion, the fact that in two of the dogs, almost full "recovery" was present as early as 35 and 39 days after the operation, a time much too brief to allow for adequate sciatic regeneration even after end-to-end suture, makes the validity of his report somewhat questionable.

The clinical value of our present method remains to be explored. The animal experiments have revealed no features that would discourage a clinical test. Local veins could perhaps serve as source of sleeves.

SUMMARY

Experiments were carried out in a total of 31 rats and 15 cats to test the practicability of bridging nerve gaps by cylindrical blood clots sheathed by sleeves of artery or collagen tubing. The gaps measured up to 3 cm. in length, i.e., thirty times the diameter of the nerve. In one series, the liquefying potency of the blood core was increased by the addition of large amounts of leukocytes (buffy coat). In other series, various types of fine artificial fibers (glass, Nylon, rayon, "Cordura") were embedded lengthwise in the blood core for purposes of consistency and orientation.

Aside from certain technical failures, excellent nerve regeneration was obtained in blood bridges without or with fiber cores; in the latter case only, if the fiber cables were firmly secured to both stumps. Functional recovery and histological appearance proved that in many cases the results were not appreciably inferior to those after end-to-end union.

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OBSERVATIONS ON ENCEPHALOGRAPHIC FINDINGS IN CEREBRAL TRAUMA

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THE AUTHORS have had opportunity for studying encephalograms done in an Army Hospital serving as a neurological and neurosurgical center over a period of nine months. The majority of cases were post-traumatic. It is believed in view of the rather limited data available in the literature on similar studies the findings at this installation are of sufficient value to be recorded.

MATERIAL STUDIED

During the period mentioned above, 261 patients were examined by lumbar encephalograms. Of these, 177 had head injury with skull defects, while 29 had closed head injuries, making a total of 206 cases, or 79 per cent. There were 32 with idiopathic epilepsy, and in 23 cases examination was made for other lesions of the central nervous system. The x-ray reports were available in all cases, and the actual films were available for review and for special measurements used in this study in 193 cases, 159 of which were cases of head injuries. In some instances the clinical records were not available at the time of study.

DESCRIPTION OF TECHNIQUE

Encephalographic procedures at this hospital have been carried out entirely in the radiological department.

Examinations were done in the afternoon. The patient was prepared by omitting the noon meal and giving pre-anesthetic medication in appropriate dosage one to two hours prior to the procedure. Premedication usually consisted of morphine, gr. $\frac{1}{6}$ and scopolamine, gr. 1/150. All encephalograms were done under intravenous anesthesia. Sodium pentobarbital ($2\frac{1}{2}$ per cent solution) was used,[6] the total dose varying with the individual.

When ready for the procedure, the patient was placed on a support attached to an ordinary radiographic tilting table with the table in vertical position. The support consisted of a board fastened to metal arms which could be attached to the table in the manner of the usual foot-board. The patient was arranged in the sitting position with his left side against the surface of the table, arms extended and resting on a small bedside table, which could be adjusted to the proper height. The head was placed with the chin resting on a U-shaped support on the bedside table.

The two-needle method of drainage of the cerebrospinal fluid was used, one needle being inserted between the 2nd and 3rd lumbar vertebrae and the second one between the 3rd and 4th lumbar vertebrae. The fluid was permitted to escape freely, each 20 cc. of fluid being replaced with 20 cc. of air. As much fluid was removed as possible, usually 100 to 150 cc.

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being withdrawn. It was not felt necessary to resort to manipulation of the head. This method was used because it was believed that a freer interchange of fluid and air was possible and that more complete and somewhat quicker drainage could be obtained.

After drainage was completed, anesthesia was discontinued, the table was returned to horizontal position, the support removed and radiographs were taken immediately.

In all cases a complete series of roentgenograms was made, consisting of anteroposterior, posteroanterior, and both lateral stereoscopic views. Except in unusual cases where it was felt that further information might be obtained, the vertical position was not used. During the taking of the films a standard head-support attached to the radiographic table was employed to maintain position and fixation of the head. Wedge-shaped supports were used beneath both shoulders for the PA view and beneath the contralateral shoulder for the lateral view.

The usual radiographic technique consisted of the use of the Potter-Bucky diaphragm, intensifying screens and a target film distance of 36 inches. Radiographic factors were: Kilovoltage, 70; Milliamperes, 100. The time was varied in individual cases as indicated, the average patient requiring 0.4 second for lateral views and 0.8 second for AP and PA views.

The films were developed immediately and checked for the necessity of any further study as well as for technical quality before the patient left the department.

INDICATIONS AND CONTRAINDICATIONS

I. *Indications*

(A) Any head injury with sequelae
1. Penetrating head wound
2. Depressed fracture
3. Simple fracture
4. Post-traumatic symptoms without demonstrable injury to vault

(B) Convulsive seizures
1. Epilepsy with deterioration
2. Focal seizures
   a) Focal tracings in electroencephalogram
   b) Localizing neurological signs
3. Convulsive seizures beginning after the age of 30
4. Post-traumatic convulsive seizures

(C) Signs or symptoms of degenerative cerebral disease

(D) Suspicion of brain tumor without signs of increased intracranial pressure

II. *Contraindications*

(A) Absolute—presence of choked disc

(B) Usually—other clinical or x-ray signs of increased intracranial pressure

METHOD OF MEASUREMENT

Early in the course of this study it became obvious that some objective method of measurement of ventricular size would be useful. It was felt that measurement would (1) aid in obviating the factor of personal judgement in analyzing encephalograms for evidence of dilatation, (2) form a basis of comparison between cases, and (3) provide comparison between repeat examinations in the same case.

Various measurements were considered, but the most satisfactory standard for our purpose was found to be the size of the body of the lateral ventricle on the AP view from the superior medial angle to the closest point on the inferolateral walls of the ventricle. A similar measurement was used by Paul and Erickson in a study recently reported. This measurement was recorded on a series of 168 encephalograms with satisfactory filling of the ventricles and subsequently correlated in the individual cases with the impression as to the size of the ventricles. Establishment of a norm is difficult, but taking normal controls and the clinical picture into consideration, the following limits were established and found to be helpful during the course of the study.

(1) 1.1 cm. and below, definitely normal
(2) 1.2 cm. to 1.4 cm., usually normal
(3) 1.5 cm., borderline
(4) 1.6 cm. and above, definitely dilated.

Two other measurements were considered and used in some cases. One was the measurement of the width of the upper border of the lateral ventricle from the medial to the lateral angle. It was found that this compared favorably in accuracy with that described above but ordinarily gave no additional information. The third was an attempt to measure the width of the anterior horns, and was the distance from the superior medial angle of the lateral ventricle to the lateral wall of the anterior horn taken at approximately a 30° angle from the horizontal. It was found to be less reliable than the other measurements but served its purpose well in comparison of first- and second-day films, which is being considered in a separate study.

It was realized that validity of such measurements might be questioned on the basis of change in position of the head or the x-ray tube during the taking of roentgenograms, particularly since in many cases they were used for comparison between the first- and second-day films. In order to determine their accuracy against this factor, additional films were taken in several cases, first varying the degree of tilting of the tube up to 10° from the usual angle in both directions, second with slight rotation of the head in both directions and third, comparative studies on stereoscopic pairs. In no instance was the variation more than 0.2 cm. from film to film of the same case taken on the same day. Reexamination in two cases done several weeks apart showed no greater variation from the original figures.

The faults inherent in any method of measurement of the ventricular system are well recognized; nevertheless some standard would appear to be desirable. It is believed that the method used is satisfactory because: (1) the measurements are easily obtained from a single film, (2) they can be reproduced in the same patient with a standard technique, (3) they are sufficiently reliable for ordinary purposes, (4) they measure the width of the body rather than one of the horns of the ventricle, and (5) they are more dependable than other measurements in the presence of minor deviations from the standard projection.

FAILURE OF VENTRICLES TO FILL

Failure of the ventricles to fill is not necessarily an indication of pathology. Lemere and Barnacle,[9] in reviewing 800 encephalograms, found that the ventricles did not fill in 8 per cent of their cases. Mayfield and Bell[10] place the percentage of nonfilling at 4–8 per cent.

In the series of 261 encephalograms reviewed here, the cases are divided into three groups: those showing satisfactory filling, poor filling, and no filling. Satisfactory filling was shown in 227 (87.5 per cent), poor filling in 15 (5.5 per cent), and no ventricular filling was revealed in 19 (7 per cent). The distribution of the type of filling among the various groups studied is shown in Table 1.

TABLE 1

Type of ventricular filling in 261 encephalograms

Type of Cases	Satisfactory Filling	Poor Filling	No Filling	Total
Head injury with skull defect	160	8	9	177
Head injury without skull defect	25	1	3	29
Idiopathic epilepsy	27	3	2	32
Others	15	3	5	23
TOTAL	227	15	19	261

The reasons for the failure of the ventricles to fill are unknown. On many occasions a second attempt at encephalography, carried out under precisely the same conditions as the first, will produce satisfactory filling. Grant[7] stresses that complete replacement of cerebrospinal fluid by air should be attempted and such replacement was attempted in all cases in this series.

In an attempt to correlate the adequacy of filling of the ventricular system with the amount of fluid replaced, the cases were divided into those in which over 150 cc. of fluid had been replaced and those in which replacement had been less than 150 cc. There were 142 case records available for this portion of the analysis. In 58 the amount of replacement was 150 cc. or more: 55 (95 per cent) showed satisfactory filling, 1 (2 per cent) poor filling, and 2 (3 per cent) no filling. Of the 84 cases in which replacement of fluid was less than 150 cc., 71 (84 per cent) revealed satisfactory filling, 4 (5 per cent) poor filling, and 9 (11 per cent) no filling. It would thus seem that the amount of replacement of cerebrospinal fluid by air has a definite relationship to the completeness of the filling of the ventricles. However, it is possible that ventricles markedly dilated as a result of cerebral atrophy fill more easily and completely than do normal or less markedly dilated ventricles.

Because of the transfer of patients to other installations with consequent loss of their records, it was impossible to further analyze the failure of the ventricles to fill, but it is stressed that in every case an attempt was made to completely drain the cerebrospinal fluid.

NORMAL AND ABNORMAL VENTRICULAR SYSTEMS

In 168 cases of all types the ventricular filling was satisfactory and the films were available for review. The encephalograms were abnormal in 117 cases and normal in 51. The distribution of normal and abnormal encephalograms among the types of cases studied is shown in Table 2.

TABLE 2

Distribution of normal and abnormal encephalograms in 168 cases of satisfactory ventricular filling

Type of Cases	Normal	Abnormal	Total
Head injury with skull defect	34	96	130
Head injury without skull defect	4	9	13
Idiopathic epilepsy	7	4	11
Others	6	8	14
TOTAL	51	117	168

Shift of the Ventricular System

Of the 143 cases of head injury in which filling of the ventricles was satisfactory and the films and clinical records were available, 17 (12 per cent) showed a shift of the ventricular system towards the side of the cranial defect. Hauptmann,[8] Travers[12] and others attribute this shift entirely to cortical fibrosis. Crothers and Wyatt,[2] in reviewing 1700 encephalograms performed on children with atrophic lesions, noted the frequent shift of the ventricles toward the atrophic side and felt that the mechanism was a pushing over of the growing normal hemisphere to the atrophic side.

Considering the short time elapsed between injury and examination in most of the cases reviewed here, it seems unlikely that fibrosis played a significant role in the displacement. It is believed that a shifting and expanding of the remaining brain helped occupy the space resulting from extensive loss of cerebral substance and was the cause of the shift of the ventricles.

Symmetrical Enlargement of the Lateral Ventricle and the Third Ventricle

In 12 (8.5 per cent) of the 143 cases of cranial trauma there was symmetrical enlargement of the lateral and third ventricle. Two patients with epilepsy (not traumatic) also showed this type of ventricular dilatation. The mechanism of such dilatation is debatable, but it seems most likely that in the majority of cases it is due to generalized loss of cerebral substance with secondary dilatation of the cerebrospinal system.

However, in some cases of cerebral trauma where diffuse bleeding throughout the subarachnoidal system undoubtedly occurred, with secondary fibrosis, the factor of deficient absorption of fluid must be considered. In the majority of cases of open head injury the subarachnoidal markings were absent around the defect, indicating that at least a portion of the

absorbing mechanism was not functioning. If this obliteration of the subarachnoidal spaces were sufficiently wide-spread, the factor of obstruction with consequent internal hydrocephalus would be an important one. The fourth ventricle would also be dilated, but, unfortunately, by any routine encephalographic procedure the fourth ventricle is not well visualized.

The findings and cases reviewed here would indicate that while the partial obliteration of the subarachnoidal spaces with consequent deficiency of cerebrospinal-fluid absorption may play some part in the symmetrical ventricular dilatation, the principal etiology of such dilatation is diffuse loss of cerebral substance. In agreement with this conclusion are the findings in two cases of epilepsy showing such enlargement in conjunction with normal subarachnoid patterns.

Symmetrical Enlargement of the Lateral Ventricle without Enlargement of the Third Ventricle

Eleven (8 per cent) of the 143 cases of cranial trauma revealed enlargement of both lateral ventricles without enlargement of the third ventricle. The underlying mechanism of such dilatation is most likely diffuse cerebral atrophy secondary to trauma.

Bilateral Asymmetrical Enlargement of the Lateral Ventricles

In 41 (29 per cent) of the 143 cases of cranial trauma bilateral asymmetrical enlargement of the lateral ventricles was evident. Of these, 31 (75 per cent) also showed dilatation of the third ventricle, whereas in the remaining 10 (25 per cent) the third ventricle seemed normal. The side of the greatest enlargement of the lateral ventricle was always the side of the injury in both open and closed head injuries.

Unilateral Enlargement of the Lateral Ventricle

In 42 (29 per cent) of the 143 cases of cranial trauma there was enlargement of only one lateral ventricle. The mechanism of such enlargement is most likely direct loss of cerebral substance without diffuse cerebral change.

Second-Day Encephalographic Studies

In 60 cases the appearance of the ventricles was checked on the day following the encephalography.* These follow-up studies proved interesting. They showed some enlargement of the ventricles in more than one-third of the cases. The enlargement was marked at times. Some porencephalic cysts, not visible during the first day examination, became visible on the later films. At times a peculiar bulging of the interventricular septum was found on the second day. These studies were of theoretical and practical interest. The existing brain atrophy was at times much more obvious on the second

* Schatzki, R., Baxter, D. H., and Troland, C. E. Second-day encephalography with particular emphasis on size of ventricles. (To be published)

day and the differential diagnosis between a space-occupying process and atrophy was facilitated in some cases by the follow-up study.

CORRELATION BETWEEN TIME OF INJURY AND VENTRICULAR ENLARGEMENT

There were 126 cases of cranial trauma, both cranial defects and closed head injuries, divided into groups according to length of time between time of injury and encephalography. Only those cases were reviewed in which the exact time of injury was known and the encephalographic pictures were excellent. In 33 (26 per cent) injury had been sustained less than three months before encephalography and of these, 20 (60 per cent) showed ventricular enlargement of some type.

Mayfield and Bell[10] and others state that shrinkage of the brain following injury is not demonstrable for three to four months after the injury. The mechanism of the shrinkage is not definitely known. It might be due to impaired circulation or to fibrosis and contracture following injury, and it is probable that both of these factors play some part in ventricular dilatation at the injury. However, the principal mechanism is probably simple loss of brain substance, perhaps mainly lipoids. This fact is indicated by the early dilatation (less than three months) in 60 per cent of the cases available.

We believe that ventricular enlargement can be demonstrated in a majority of cases within a very short time after injury, that is, as soon as cerebral edema consequent upon the injury has subsided.

CORRELATION OF NEUROLOGICAL FINDINGS AND ENCEPHALOGRAMS

Of 129 cases of cranial trauma in which filling of the ventricles was satisfactory and there was adequate clinical information on the neurological side, 11 (8.5 per cent) revealed a normal ventricular system in the presence of clinical evidence of definite neurological damage. In 61 (47.5 per cent) there was positive evidence of neurological damage and abnormal encephalogram while 26 (20 per cent) had no clinical or encephalographic evidence of cerebral damage. In 31 (24 per cent) the encephalogram was abnormal although neurological examination was negative.

The neurological examination in many cases was relatively gross as it was confined to examination of the motor and sensory systems, the visual fields, reflex changes, gross dysphasia and the cranial nerves, without adequate psychiatric study or an estimation of any mental retardation. If these two studies had been carried out it is believed that the percentage of cases showing clinical evidence of cerebral damage would be somewhat higher than the above figures would indicate.

The clinical record in many cases, particularly as concerned neurological status soon after injury, was too inadequate for a statistical correlation of neurological and encephalographic findings but certain broad impressions and findings could be gathered. For the most part the encephalogram and the clinical findings were very closely related. This was particularly true in cases of unilateral ventricular dilatation where the cerebral loss was rela-

tively circumscribed and the clinical picture was that of localized cerebral damage.

If the occipital or temporal regions showed dilatation there were field defects of varying sizes, and if the cerebral loss was mainly in the Rolandic region there were varying degrees of motor paralysis. Few patients, however, showed persistent sensory defect although there was encephalographic evidence of severe damage to the sensory cortex. Sensation in the great majority of cases had been largely taken over by the opposite cortex or lower centers.

The degree of motor paralysis and spasticity varied greatly in patients showing essentially the same encephalographic picture. It had been hoped that it might be possible to more closely correlate the degree of spasticity with the encephalographic picture but this was not possible because of lack of sufficient data. The impression was gained, however, that spasticity was most pronounced in those cases showing cerebral damage anterior to the Rolandic area along with damage to the motor area.

In some cases there was considerable variation in the clinical picture in the presence of essentially similar encephalographic findings. This was particularly true in cases of extensive generalized ventricular dilatation, some showing definite evidence of cerebral impairment while in others the changes were very meager.

POST-TRAUMATIC SYNDROME

In 123 cases the encephalographic fillings were satisfactory and information was available as to post-traumatic symptoms. In this group 116 patients had cranial defects, and 7 had closed head injuries. The number of closed head injuries in this series is of course too small for a statistical study. Six of the 7 patients had severe post-concussion syndromes consisting of headache, dizziness, intolerance to alcohol and some memory loss. Four of these 6 had normal encephalograms. Of the 116 patients with cranial defects, 47 (41 per cent) had post-traumatic symptoms (headaches, dizziness, etc): 14 (29 per cent) of these had normal encephalograms. There were 69 (59 per cent) who had no post-traumatic symptoms.

The post-traumatic syndrome in cases of cranial defects was almost always very mild, usually consisting of a slight headache. In the closed head injuries the syndrome was always very pronounced, but no conclusions can be drawn from this fact as only the more severe closed head injuries are seen in a general hospital and only a small percentage of these cases are submitted for encephalography.

The post-traumatic syndrome has been regarded by many as a purely psychiatric manifestation. However, Mayfield and Bell,[10] Friedman,[5] Travers,[12] Hauptmann[8] and others have shown definite encephalographic changes following closed head injuries in patients having post-traumatic symptoms but no localizing neurological signs. Their findings and the few cases in this series would indicate that encephalography should be used more widely

in post-traumatic cases, in order to ascertain the presence of definite cerebral damage. A normal encephalogram does not necessarily rule out organic brain damage but it does indicate that such damage is probably not severe.

Encephalography has been reported by many authors to give relief of post-traumatic symptoms but in this series the amount of relief was meager and minimal.

SUMMARY

1. The encephalographic findings in a series of 261 patients, 206 of whom had had cranial trauma, have been reviewed. Of these, 177 had head injuries with skull defects while 29 had closed head injuries.

2. Abnormal encephalograms were found in 74 per cent of the cases of head trauma.

3. Unilateral dilatation of the ventricles was found in 29 per cent of the cases of head trauma. Symmetrical bilateral dilatation of the ventricles was found in 16 per cent of these cases; asymmetrical bilateral dilatation in 29 per cent.

4. In a group of 60 cases in which second-day study of the ventricles was made, an increase in the ventricular size on the second day was found in over one-third.

5. Many of the encephalographic abnormalities were found within a short time after injury.

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SEPARATION AT THE SUTURE SITE AS A CAUSE OF FAILURE IN REGENERATION OF PERIPHERAL NERVES

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COMPLETE failure of regeneration of a nerve following satisfactory suture is unusual and, except for the relatively few instances of ischemic and traction palsy, has been found to be due to separation of the anastomosis. This should not be misconstrued to mean failure to attain return of complete function, since this is obviously more complicated and not uncommon except in nerves distributing gross function, such as the radial and sciatic.

During the 6 months from March 1 to September 1, 1945, 605 nerve sutures were studied at a neurosurgical center in an Army General Hospital (see Table 1). Of these, 148 nerves had already been sutured in overseas

TABLE 1
Distribution of 605 nerve sutures

Nerve	Number Sutured	Suture Line Separations
Ulnar	173	1
Median	100	4
Sciatic	98	21
Radial	84	2
Peroneal	55	13
Posterior tibial	40	3
Brachial plexus	23	0
Musculocutaneous	11	0
Facial	5	0
Femoral	4	0
Miscellaneous	12	0

neurosurgical centers; the remaining 457 were sutured at this hospital during the 6-month period. All of the patients have been followed over 6 months and many over a year since operation.

The nerve sutures represent the work of many surgeons in several neurosurgical centers. The well established fundamental principles of peripheral nerve surgery were carried out by all surgeons with certain minor modifications, principally surrounding the use of tantalum. Since it is generally accepted that early nerve suture facilitates regeneration of function, it is important to recognize as early as possible any factors interfering with regeneration so that appropriate steps may be taken for correction. The clinical

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signs accepted as evidence of regeneration have been (1) a steadily advancing Tinel's sign, (2) return of gross motor function and (3) return of deep sensation into the autonomous zone. Anticipation and corroboration by changes in electric muscle reactions and electric skin resistance tests were used routinely in each case.

It can be appreciated quite readily that to wait for the return of clinical signs requires costly months. The use of fine tantalum wire for peripheral nerve sutures introduced several advantages, not the least of which is its

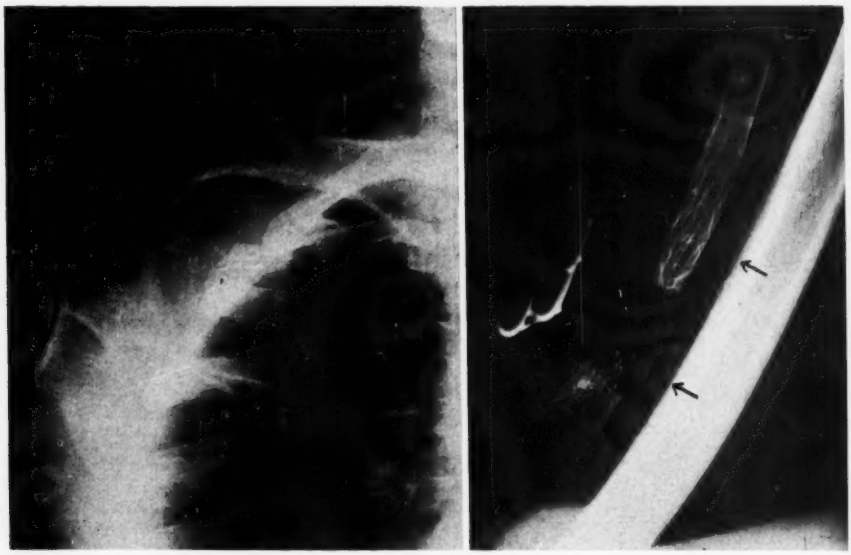

FIG. 1 (*left*). Well clustered tantalum sutures of a brachial plexus repair are visible just below the clavicle in this x-ray film taken 6 weeks after operation. No evidence of separation.

FIG. 2 (*right*). Disruption of this sciatic nerve is demonstrated by separation of the epineurial sutures (see arrows), a broken stay suture, and fragmentation with collapse of a large tantalum foil sleeve.

opacity to x-ray (Fig. 1). Early in its use it was found that rupture of the suture site could be readily detected by x-ray since part of the epineurial sutures remained attached to each segment of the disrupted nerve, presenting two distinct clusters of sutures. This led to routine postoperative x-ray examination on every nerve suture shortly after the cast was removed and mobilization of the extremity initiated. Should any slip occur in applying the cast at time of surgery, films are taken immediately. By this procedure, separation at the suture site was recognized months before failure of regeneration would have become manifest clinically. The suture line disruptions where tantalum was employed were recognized at an average time interval of 2.8 months after suture, while those done by non-opaque suture were not recognized until 7.8 months postoperatively on an average. In this

connection, it should be noted also that 77 per cent of the nerve disruptions where tantalum was used were identified first by x-ray, while in all of those where non-opaque material was used, recognition had to await clinical appraisal. Patients admitted following surgery elsewhere frequently had not had the integrity of the suture line checked by x-ray previously and suspicions of a ruptured suture line from unsatisfactory clinical progress were borne out by x-ray (Fig. 2). These instances were largely responsible for the above average being as high as 2.8 months.

Of all nerve sutures in this group 44 (7.5 per cent) suffered separation at the anastomotic site. This figure is not startling in itself and probably represents a fairly low percentage in comparison with other series, but upon further analysis of Table 1, it becomes a cause for concern to find that 34 or 77 per cent of all suture line separations occurred in either the sciatic or peroneal nerves. This gives an amazingly high incidence of about 22 per cent failure in sutures in each of the two nerves, solely from this cause.

There are certain factors which may (I) predispose a suture site to separation and (II) be a direct cause of rupture of a nerve anastomosis.

I. *Predisposing Factors in Separation at Suture Site.* The principal factors are those that influence the size of the gap to be overcome in the nerve repair. It must be that every nerve has a practical limit to which it may be stretched and still function, although the ingenuity of surgeons, past and present, has successfully overcome amazing gaps in certain instances. This limit, however, may be altered considerably by the following factors:

(1) The variation between individuals is evident both in regard to size and nature of the normal tissues, some showing more elasticity than others, with differences in their relative disposition.

(2) Pathologic findings may limit the gap that can be overcome. The extensive intrinsic and extrinsic fibrosis and fixation, from destruction secondary to high explosives, infection and vascular damage, restrict the elasticity much more than conditions found following cutting wounds.

(3) Surgical factors restricting mobilization, such as relation of nerve branches, blood supply, important soft tissues and particularly bone structures, also limit the size of a gap that can be overcome with practical success. Strangely enough excessive gaps closed by heroic and often impractical attempts were responsible for only 4 of the 44 suture line disruptions in this series. Secondary repair here, however, is not usually rewarded by success. The average gap that had been overcome in the nerves that showed disruption was 6.2 cm., which fact in itself indicates that other factors were usually responsible. Furthermore, the final gap present when the suture disruption was repaired averaged only 7.1 cm. and in most instances could be repaired satisfactorily.

(4) Time may be a predisposing factor affecting the success of a suture, preoperatively or postoperatively. After 3 weeks following injury, in cases of wounds from high velocity missiles, it is felt that the earlier the suture is performed the less fibrous restriction is encountered.

The interval between suture and extension of the immobilized joint is quite variable and in general must be based on the individual case. Two approaches have been used with success, but attempts to compromise and combine the two have been responsible for several of the failures in this series.

(a) The first method is the use of a stay or sling suture and mobilization with fairly rapid extension of the flexed joint as early as 2 weeks after operation. This takes advantage of the ability at this time to disperse the tension over a greater extent of the nerve before it becomes fixed to the surrounding tissue by scar. This method is particularly applicable when the repair has been relatively early.

(b) The second method is without the use of a stay suture. Here the immobilized joints must remain fixed 2 to 3 weeks longer and extension be more gradually carried out over a longer period. After a certain interval, rupture of the suture site will not occur, but if the stress is too great the more hopeless situation of intermittent rupture of the individual fascicles over a longer segment of the nerve results—giving the so-called "stretch" or "traction palsy." This may or may not be associated with complete loss in continuity of the nerve—this is unimportant, however, from a physiological standpoint since the paralysis is complete and frequently does not lend itself to successful surgical repair.

(5) Sutures.* At no instance could the rupture of the anastomosis be found attributable to the suture material—unless perchance a stay suture had been employed (Fig. 2). In all cases in which the metallic sutures were used in the epineurium alone, it can be assumed that they pulled out of the end to which they were less firmly attached. The epineurium on each segment of the sutured nerve is of about the same quality and since it is a most unusual technique that can encompass identical margins in passing the suture through the epineurium of each segment of the nerve, it is a fortunate happenstance that a nearly equal number of sutures have remained attached to each end in many of the cases reviewed. Frequently, to avoid the element of chance, a single epineurial suture has been taken at a measured distance from the suture line on each segment (Fig. 3). This method has been followed in every instance at this hospital where the suture was performed with non-opaque suture material. This situation obtained in several cases when the supply of tantalum was temporarily depleted.

The use of a "stay" or "sling" suture of a heavier tantalum or silk as an additional factor preventing separation of the suture line is probably more

* The strength on straight pull of tantalum is about the equivalent of braided silk of a similar diameter. Samples of tantalum of 2.9 mil diameter measured a tensile strength of 0.74 pound while those of a slightly larger braided silk of 3.5 mil diameter tested at 0.79 pound. An aggregate of many samples of varying size averaged 88,000 straight pounds per square inch for tantalum and 82,000 for silk. This may be further modified by the fact that while tantalum is unaffected by sterilizing, silk loses about 10% of its tensile strength. Furthermore, the knot pull value of tantalum is about the same as the straight pull. Indeed, the tied tantalum sutures often break away from and not at the knot, while the other non-metallic suture materials lose about 30% strength on knot pull.

efficacious than the statistics in this series would indicate. Nine of the 44 ruptures had been repaired with the support of a stay suture. These sutures usually displayed a break in the stay suture itself (Figs. 2 and 4). It is quite evident that stay sutures will not prevent disruption of the suture line. However, in several of these cases the use of a stay suture was the result of the presence of unusual tension at the suture line. Furthermore, they may have been quite instrumental in preventing mishap in other instances, particularly in those cases where extension is started early. The use of a stay suture passing directly through the nerve itself is not an ideal procedure from a pathological standpoint and may destroy a few fascicles. However, the many excellent results now seen following its use can refute much criticism.

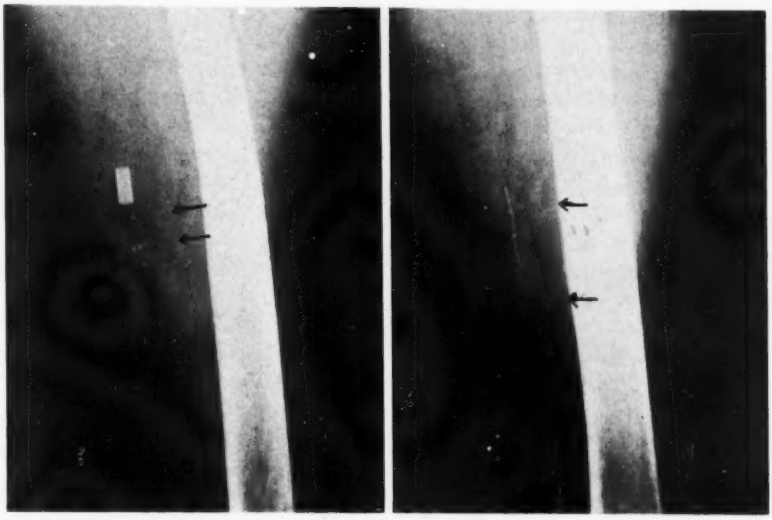

FIG. 3 (*left*). Single metallic sutures placed 1½ cm. on each side of the suture line as landmarks (one lies just in the end of the metal cuff) give added evidence of rupture in this peroneal portion of the sciatic nerve.

FIG. 4 (*right*). Arrows point to separate clusters of fine tantalum epineurial sutures in the two ends of a sciatic nerve which ruptured despite the presence of a stay suture.

II. *Direct Causes of Disruption of Suture Site.*

1. Inadequate mobilization of the nerve to be repaired is probably the most common cause for suture line disruptions and was felt to be responsible for 14 of the cases in this review. Several of the suture line disruptions have been found beneath short and inadequate incisions. Sufficient mobilization with dissection and preservation of the nerve branches and vascular supply is necessary to promote a successful suture without undue tension in most cases of war injuries. This is a fundamental of peripheral nerve surgery which

has been frequently neglected in the instances of rupture of the nerve suture.

2. The failure to maintain positioning in the postoperative period was the probable cause for 12 suture line disruptions. This group includes such accidents as losing position while applying plaster, changing a cast, removing sutures, broken or inadequate splints, etc. One uncooperative patient removed his plaster on the first postoperative day, resulting in a disruption which demanded immediate repair. Another patient fell while learning to use crutches immediately after the removal of plaster. Two patients flexed their hips when their spica cast was bivalved for removal of skin sutures. One soldier with a median nerve suture had his plaster splint removed on the 16th postoperative day by mistake. He expressed his freedom immediately with a workout on a punching bag. The result was seen in two separated rows of tantalum sutures. The second repair of this nerve likewise ruptured, requiring a third suture which at this time is showing satisfactory evidence of regeneration. In other cases, too rapid or forceful extension of the flexed joint after the removal of plaster was felt to be the most likely factor in disruption. Some of the peroneal suture line disruptions occurred in cases in which there had been gradual extension by means of a turnbuckle incorporated at the knee, although some have recommended this system as ideal. In some instances where the sciatic nerve or its branches were involved, rupture of the suture line occurred while the plaster cast was being applied. This was proven in several cases by immediate x-ray examination. This occurrence undoubtedly resulted when the surgeon, fatigued by the prolonged operation, left the application of plaster to others who did not appreciate as much the importance of maintaining the position of the extremity. One may be assured that after such an experience, the operator will not fail to maintain the desired position of the extremity until the plaster is applied. The use of a preformed bivalved spica cast, as urged by Spurling, provides good assurance against such mishaps.

In many instances of disruption, however, no reasonable cause or time of separation at the suture site can be offered. It is interesting to note that while many neurorrhaphies involve gaps bordering on the impossible, comparable gaps have been successfully overcome. On the other hand, many disruptions occurred in relatively small gaps where the suture was relaxed but where the surgery was inadequate.

MECHANICAL SUSCEPTIBILITY OF CERTAIN NERVES

The fact that 34, or 77 per cent, of all suture line disruptions occurred in the sciatic and common peroneal nerve demands further explanation for this particular susceptibility. Investigation of this group reveals that in all except 3 cases the peroneal nerve or peroneal component of the sciatic nerve was involved. (In those 3 the posterior or tibial half of the sciatic alone was involved.) It is therefore seen that the peroneal nerve is directly or indirectly

a party to 70 per cent of all suture line disruptions. Additional evidence indicting the peroneal nerve, was the fact that in 3 cases where the common peroneal and posterior tibial divisions were split and sutured separately with identical gaps to overcome, the peroneal half alone ruptured. This appears to indicate that disruption is more typical of the peroneal nerve and apparently for some mechanical reason. The review of several dissections revealed that the peroneal nerve, when adequately mobilized to permit suture, is the only major nerve whose line of stress is not mediated by some soft tissue at a point of flexion. Here the line of stress is a straight pull between two bony points, the head of the fibula and the pelvis. All gain has to be made through direct stretch of the nerve and there is nothing to break the pull, or any sudden jerk, but the nerve itself. This mechanical disadvantage peculiar to the peroneal nerve is felt to be the principal reason for its susceptibility. When the injury is in the region of the common peroneal nerve several centimeters can be made up with ease simply by flexing the knee without any further mobilization or relaxation. This places considerable stress on a relatively short section of nerve and may be a factor in the frequent disruption occurring in this nerve, as mentioned above in the discussion of inadequate mobilization. Another factor may be the weight of the lower extremity and relative difficulty in holding the positioning necessary for the success of the suture—especially where only a small excursion may overcome the holding power of the anastomosis if the nerve be sutured with some tension. It should be emphasized again that unless a stay suture is used, nerve anastomosis is done delicately for means of careful approximation only and not to withstand tension.

Although the various biochemical determinations have not been carried out on any of the patients of this series, there has been no gross evidence of the presence of a nutritional deficiency that might be a factor in the patients who were reviewed.

In 3 cases (a median, a sciatic and a peroneal), the disruptions were repaired only to suffer another disruption. In two of these the third repair already shows evidence of success. In 3 other cases attempts at repair disclosed that the new gap was too great to make further attempt at suture possible. In all cases, however, it should be urged that the ruptured suture site be explored and every possible effort be exerted to correct the misfortune.

CONCLUSIONS

1. Disruption of the suture site is the usual cause for complete failure of regeneration following nerve suture.

2. Valuable time and hence chance of recovery is saved by early recognition and repair of suture line separation.

3. Tantalum sutures do not break easily and by reason of their opacity to x-ray give a prompt and accurate estimation of the integrity of the suture site.

4. Metallic markers should be used on each segment of a sutured nerve when non-metallic material is used for anastomosis in order to detect any separation.

5. The peroneal nerve and the sciatic nerve, where the peroneal component is involved, are the nerves particularly affected in the rupture of the suture site.

6. Possible reasons for disruption and the particular susceptibility of the peroneal nerve are advanced.

CORTICAL LOCALIZATION BY ELECTROENCEPHALOGRAPHY

THE VALUE OF A QUANTITATIVE AND STATISTICAL ANALYSIS OF HOMOLOGOUS RECORDINGS OBTAINED SIMULTANEOUSLY

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FOLLOWING a trial of various reported techniques of electroencephalography and as a result of electroencephalographic experience on both neurological and neurosurgical services, we have gained the impression that the usefulness of electroencephalography as employed routinely in most techniques for purposes of localization is limited to relatively gross lesions. In the hope of improving its value, an investigation of the localizing accuracy that might be achieved by a quantitative comparison of bilateral hemispheric data, obtained by recording homologous potentials simultaneously, has been undertaken. It seemed possible that a comparison of the focal abnormal findings on one side against the normal side as a control might possess distinct advantages for purposes of localization. The principle involved is the same as that employed in the neurological examination. Isolated sensory, motor and reflex findings may be obvious when the changes are marked but an evaluation of finer differences is necessarily based upon the comparison of homologous parts.

Although the value of homologous recordings has been recognized to a certain extent in the techniques used in several laboratories, the importance of recording them simultaneously, in order to enable direct and quantitative comparison, and the necessity of obtaining a sufficient number of runs with respect to all areas to permit a statistical handling of the data, have not been fully appreciated. The importance of the statistical approach proposed can not be overemphasized as a necessary corollary to the technique of homologous recordings.

It is axiomatic in such a study that rigid criteria for the technique of electroencephalography and for the analysis of the record be established and that the correlative value of the results be determined statistically in large numbers of cases that are well controlled from a clinical standpoint. The present report, based on 1200 cases obtained at the University of California Hospital, is an initial attempt at such a correlative study.

METHODS AND TECHNIQUE

The quantitative and statistical analysis of homologous recordings obtained simultaneously constitutes the basis of the technique used in this study. The simultaneous recording of homologous runs, side by side, permits a direct comparison of bilateral hemispheric data. Variations of homolo-

gous potentials and frequency ranges may be expressed quantitatively. The degree of bilateral synchronism or asynchronism and the difference of wave form may likewise be compared directly and thus a close quantitative comparison of homologous runs may be made. Even in temporal and occipital areas, where poor synchronism and bilateral inequality of potentials are frequently found in normal individuals, changes in the frequency range and in the form of the pattern remain as criteria of abnormality. Such exceptions to the quantitative approach proposed do not invalidate the principle of the comparison of homologous recordings any more than differences in strength in a right- or left-handed person nullify the value of the motor examination.

Electrode Placement

The number and disposition of electrodes to be used in any given case logically depend upon the nature of the case and the purpose of the study. Verification of a generalized cerebral

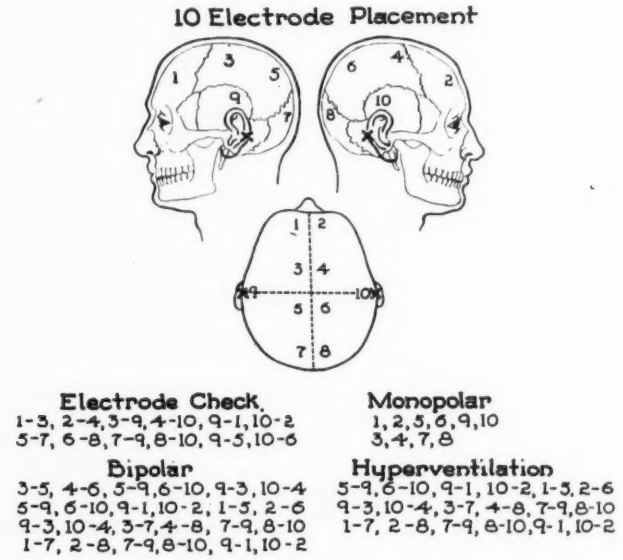
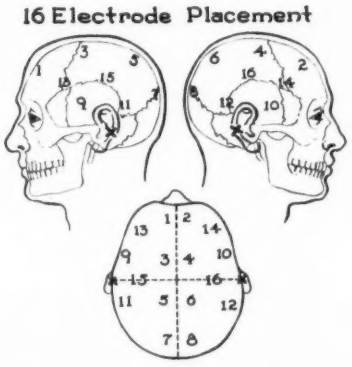


FIG. 1.

dysrhythmia in convulsive states of undetermined etiology, with either petit mal or generalized grand mal seizures and without localizing signs by neurological examination, does not require the same number of electrodes as would be the case in patients suspected of brain tumor or focal epilepsy, in whom localization is of paramount importance. The number of electrodes employed in routine screening techniques of the first type vary from 4 to 14. Despite general agreement that the electrode placement should cover both hemispheres and be symmetrically arranged with respect to homologous regions, many laboratories place the electrodes high on the scalp in rows more or less parallel to the midline, neglecting the tem-

poral lobe and other lower areas. A minimum of 8 electrodes might afford representative coverage for the available cortical areas of all cerebral lobes but because of the disproportionate size of the frontal lobes, a more even distribution of electrodes requires 10. The 10-electrode placement as used routinely in this study is shown in Fig. 1.

For a more detailed study and for purposes of localization, a spacing of electrodes not over 5 or 6 cm. apart is desirable. Adequate coverage of the available cortical areas for routine purposes may be accomplished by a 16-electrode placement as indicated in Fig. 2. Extra bilateral suboccipital electrodes have been utilized in suspected lesions of the posterior fossa.



Electrode Check

1-3, 2-4, 3-13, 4-14, 13-1, 14-2
5-7, 6-8, 7-11, 8-12, 11-5, 12-6
9-15, 10-16, 15-11, 16-12, 11-9, 12-10

Monopolar

1, 2, 15, 16, 7, 8
3, 4, 11, 12, 9, 10
5, 6, 13, 14

Bipolar

1-3, 2-6, 5-9, 6-10, 9-1, 10-2
1-7, 2-8, 7-15, 8-16, 15-1, 16-2
3-5, 4-6, 5-15, 6-16, 15-3, 16-4
3-7, 4-8, 7-11, 8-12, 11-3, 12-4
5-11, 6-12, 11-13, 12-14, 13-5, 14-6
9-13, 10-14, 13-15, 14-16, 15-9, 16-10
1-5, 2-6, 5-11, 6-12, 11-1, 12-2
3-7, 4-8, 7-9, 8-10, 9-3, 10-4
13-15, 14-16, 15-7, 16-8, 7-13, 8-14

Hyperventilation

1-5, 2-6, 5-11, 6-12, 11-1, 12-2
3-7, 4-8, 7-9, 8-10, 9-3, 10-4
13-15, 14-16, 15-7, 16-8, 7-13, 8-14

FIG. 2.

A more detailed analysis, such as may be required in special cases, of course involves the use of extra electrodes in the region to be investigated. A catheter type of nasopharyngeal electrode has been used in cases of suspected lesions in the region of the third ventricle or brain stem. The electrode routinely used in this study has been the flattened soldered disc fixed to a cotton-wrapped, multiple-strand, light, flexible cable wire.* The generally accepted practice of applying the electrode with electrode paste and collodion after cleaning the desired spot on the scalp with acetone has been followed.

* Recently we have been using needle electrodes made with #27 gauge hypodermic needles of $\frac{1}{2}$ or $\frac{3}{8}$ inch length and soldered to the same wire as used with the disc electrodes. This type of electrode saves considerable time in making the electrode placement and produces a record almost free of the troublesome electrode artifact described in this paper. Its use has caused surprisingly little discomfort to the patients but has not always been found practical for infants or small children.

Calibration

A careful calibration of the electroencephalographic equipment is obviously of paramount importance if a close quantitative comparison of homologous runs is to be made. In addition to the usual calibration with a known input (50μV), which serves as a yardstick for the measurement of potentials, all channels are closely checked under actual running conditions by recording the same electrode setting simultaneously. The precentral and occipital electrodes are usually satisfactory for this purpose inasmuch as the recordings from this setting are, as a rule, sufficiently large and regular to facilitate accurate measurement and the desired comparison of channels. All channels are calibrated by this method not only initially but before hyperventilation, at the end of the record and at any other time throughout the recording that any change in channel amplification is suspected.

Electrode Artifact

The subject of electrode artifact deserves special attention. Many laboratories replace "popping" electrodes either not at all or only occasionally, on the assumption that the

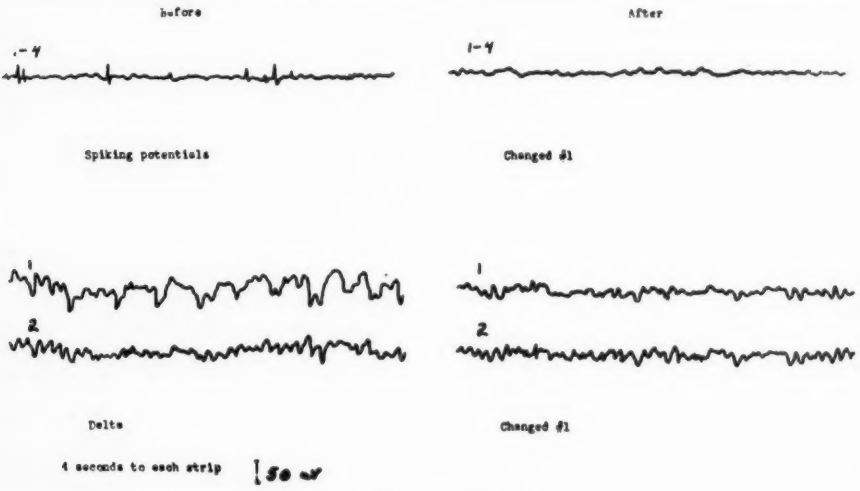


FIG. 3. Artifact.

"popping" is obvious and that the record between "pops" is satisfactory. That this is frequently not the case is illustrated in Fig. 3. Electrode artifact does not necessarily assume the "popping" form and can frequently simulate delta activity or spiking potentials (Gibbs and Gibbs[2]), and may even distort the entire run. Consequently, abnormal activity associated with possible "popping" must be checked by adjustment and, if necessary, by replacement of the electrode involved before the abnormality can be assumed to be real. In many instances it has been necessary to change the same electrode three or four times to rule out possible artifact. Although this can prove very trying and requires great patience on the part of the technician, there appears to be no satisfactory alternative. Faith in the superiority of one's electrodes, interpreting abnormal activity between "pops" or any "adjustment" of the electrode are not satisfactory substitutes for changing it completely if the questionable activity persists.

Recording Routines

It is generally agreed that the over-all period of recording should be from 20 to 30 minutes at least to insure the recording of random or episodal activity of diagnostic importance. Long

runs on the same settings, however, are not essential to achieve this end. The focal dysrhythmias associated with pathological changes in the cortex are almost constantly reflected in the electroencephalogram as opposed to the episodal activity characteristic of the generalized paroxysmal dysrhythmias. An analysis of several hundred records has shown that the characteristics of any particular recording that occur constantly may be adequately analyzed in a run of 20 or 30 seconds. A maximum number of runs, utilizing as many electrode combinations as possible, has been found necessary for the statistical analysis of the records made for purposes of localization. Also repeat runs on different channels are desirable in order to rule out possible distortion by the recording equipment and the electrode placement. Consequently, a routine involving many short runs has been established in preference to one employing fewer runs of longer duration. Since the period of recording with a routine of many short runs is essentially the same as with a routine employing fewer runs of longer duration and the total period of recording with respect to any given electrode is the same as in other methods, the modified technique proposed detects random or episodal activity as satisfactorily as the older techniques.

The schedules for the standard 10- and 16-electrode placements, adapted to both 4 and 6 channels of recording, are shown in Figs. 1 and 2. Obviously, these recording routines are not duplications of the older techniques which record "down" and "across" the head, etc. Bipolar recordings across the midline have been abandoned since they do not contribute satisfactory data for the type of analysis involved.

The combination of runs in the first two or three recordings serves to check all electrodes for artifact. In a high percentage of records more data are obtained than is indicated by the recording routines as a result of the usual necessity of repeating several runs, parts of which have been distorted by artifact. The extra runs correspondingly increase the over-all time of recording so that the routine employing 16 electrodes commonly requires more than 30 minutes.

Both monopolar and bipolar recordings have been used throughout this study. Arguments as to the superiority of one type of recording over the other seem pointless when short runs of as many electrode combinations as possible are desired for the statistical analysis of the records. Although the findings by monopolar and bipolar recordings are usually consistent, we have found that this is not invariably true. In many instances the evidence that has led to a correct interpretation has been adduced largely, or even entirely, by one or the other method of recording. In view of the fact that diagnosis by electroencephalography is at best difficult and should utilize all available evidence, the use of streamlined techniques providing limited data appears unwarranted.

Hyperventilation for 3 minutes with the electrode settings as indicated in the recording routines has been used in all electrographic studies in accordance with practice elsewhere.

Number of Channels for Recording

An even number of electroencephalographic channels is required for the simultaneous recording of homologous runs. Although the study may be conducted with the use of 2 channels, the routine is greatly facilitated by 4 channels. The accumulation of the data that are essential for statistical analysis is accomplished even more readily by 6 channels. This number also allows some margin of safety over the 4-channel routine in case technical difficulties should be encountered in any of the channels of amplification or recording. Six channels, furthermore, make possible the bilateral triangulation runs shown in Fig. 2, which afford greater cortical coverage at all times throughout the recording and facilitate localization of either electrode artifact or abnormal brain potentials.

Analysis of Records

The analysis of records has been facilitated by the use of a Work Sheet (Fig. 4) which consists of a Measurement Chart, Graph and Report Form.

Measurement Chart. Homologous recordings are listed side by side in numerical order as shown on the Work Sheet. To simplify the evaluation of the bilateral synchronism of homologous runs, good synchronism is represented by the letter "S," asynchronism is indicated by

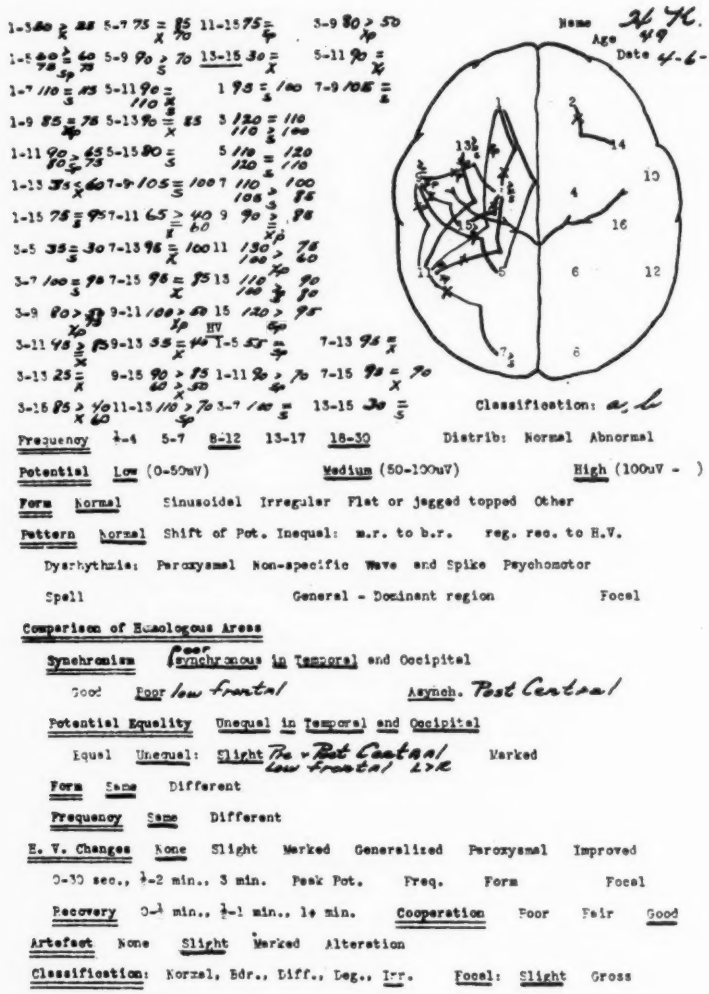

Name H. K.
Age 49
Date 4-6-42
Classification: a, L
Frequency 8-12
Distrib: Normal
Potential Low (0-50uV) Medium (50-100uV) High (100uV -)
Form Normal Sinusoidal Irregular Flat or jagged topped Other
Pattern Normal Shift of Pot. Inequal: m.r. to b.r. reg. rec. to H.V.
Dysrhythmia: Paroxysmal Non-specific Wave and Spike Psychomotor
Spell General - Dominant region Focal
Comparison of Homologous Areas
Synchronism Poor synchronous in Temporal and Occipital
Good Poor low frontal Asynch. Post Central
Potential Equality Unequal in Temporal and Occipital
Equal Unequal: Slight Marked
Post Central > low frontal L > R
Form Same Different
Frequency Same Different
E. V. Changes None Slight Marked Generalized Paroxysmal Improved
0-30 sec., ½-2 min., 3 min. Peak Pot. Freq. Form Focal
Recovery 0-½ min., ½-1 min., 1+ min. Cooperation Poor Fair Good
Artefact None Slight Marked Alteration
Classification: Normal, Bdr., Diff., Deg., Irr. Focal: Slight Gross

FIG. 4. Work Sheet. H. K. 4-6-42.

"X," and "fair" and "poor" synchronism are indicated by the symbols "Sp" and "Xp," respectively.

The comparative size of homologous potentials is based upon an over-all estimation of the runs under consideration and is indicated by the mathematical symbols for equality and inequality (=, > and <). Both symbols of equality and inequality are used for slight or questionable inequalities of homologous potentials, such as consistent differences within the range of error of the recording equipment, differences limited to the larger waves of each run or inequality of potentials in a single tracing of homologous runs, which is not confirmed on repetition. Peak potentials are measured, using a microvoltage scale based on preliminary calibration. Since the measurements are of relative value only, they are taken for the sake

of convenience from crest to trough of the waves showing the highest potentials in the run. Large abnormal waves of slow frequency are measured from peak to trough regardless of superimposed waves of smaller potential. With occasional exceptions, such peak potentials afford an index of the range of potentials attained in each run and have been found to reflect the over-all estimate of the comparative sizes of homologous runs. Furthermore, in repeated tracings the relative difference in the size of homologous runs, as indicated by the peak potentials, and to a less extent the absolute measurement of peak potentials themselves, are surprisingly constant. Although more accurate measurements based on the average potential of each run might be established, they appear neither practical for routine analysis nor necessary for the approximate quantitative evaluation of relative sizes of homologous runs.

Homologous runs are also studied for variations in their ranges of frequency and form, which, if found, are indicated beside the charted run. The presence of waves of abnormally slow frequency is represented by "Δ^{3-5}," the accompanying numbers showing the frequency range. Spiking potentials are represented by the symbol "$\sim\!\wedge\!\sim$" and when desired, the peak potentials of both the abnormally slow and fast waves can be specifically indicated "Δ^{150} and $\sim\!\wedge\!\sim^{125}$." The duration, characteristic wave form, potentials and frequency ranges of transient episodal activity which is bilaterally synchronous and equal and which involves a series of homologous runs recorded simultaneously, can likewise be conveniently indicated.

Graph. The diagrammatical representation of the unequal, asynchronous and abnormal potentials is of considerable value in that it affords a quick method for the statistical evaluation of the data. In this respect it is analogous to a spot graph, indicating variants that may be disregarded safely and focusing attention on the abnormal region. Unequal homologous potentials are plotted in their proper location on the head diagram. In the case of monopolar recordings, equal as well as unequal potentials are indicated if poorly synchronous or asynchronous, the symbols being placed beside the figures indicating the electrodes to which they apply. Asynchronous bipolar runs may be indicated by writing the appropriate symbols on lines connecting the electrodes involved. A relative hyperpotentia by bipolar recording may be conveniently represented by V-shaped lines drawn between the two electrodes, with the angle pointing to the opposite hemisphere of lower potential. Although straight lines bearing asynchronous symbols might be used for bipolar runs of equal potential, it is usually less confusing to omit these, bipolar asynchronism being readily perceived from the chart. Abnormally slow activity, spiking potentials and variations of frequency can also be indicated by their respective symbols in the same manner as bilateral asynchronism. Ordinarily, however, this is not necessary and should be omitted when possible to avoid confusion.

Report Form. The analysis may be summarized in outline form with sections devoted to the general characteristics of the record, a comparison of homologous areas, the results of hyperventilation and the final opinion. The opinion is written as a summary and is used as a clinical report to be filed in the patient's chart or sent to the referring doctor. The items listed in the outline have been carefully selected as categories in a punch-card system* to facilitate statistical analysis and cross correlation for purposes of review and research.

A systematic and detailed analysis of records may be made fairly rapidly by this method. If the peak potentials and over-all estimation of the equality and synchronism of homologous recordings are measured ahead by a trained technician, the 10-electrode test rarely takes over 10 minutes for the review and analysis and the 16-electrode studies as a rule may be completed within 20 minutes. In the course of such an analysis thorough familiarity with the record, its rhythms, characteristics and peculiarities is achieved. The importance of the outline form lies in the fact that it materially contributes to a more complete and efficient analysis of the record and lends itself to a proper organization of the data for purposes of statistical review.

PRINCIPLES OF EVALUATION

In addition to the usual electroencephalographic analysis based upon a study of the form, size, frequency and general pattern of the brain waves

* E-Z Sort Systems, Ltd., 45 Second Street, San Francisco, California.

and localization by means of triangulation, the technique used in this study provides data that require special consideration for their evaluation. Asynchronous hemispheric discharge, inequalities of homologous potentials and relative differences in the form and frequency ranges of bilateral tracings recorded simultaneously, constitute valuable evidence of abnormality. Criteria for the interpretation of such quantitative data are:

1. The inequality and asynchronism of homologous potentials primarily are of localizing value, although alone do not necessarily reveal the side of the lesion. The latter information usually is indicated by a relative irregularity of form, slower frequency range, or both, in the region and on the side involved.

2. The consistent absence of bilateral synchronism and equality of potentials in homologous runs, other than the temporal and occipital, constitute evidence of abnormality. When found alone they may be regarded as presumptive evidence of abnormality; when observed in association with other signs of abnormality, they are of corroborative value. Inequalities of homologous potentials that are synchronous should be considered as suggestive evidence of abnormality, except in the temporal and occipital regions, while bilateral asynchronism of equal potentials must be regarded as of questionable import.

3. A consistent hypopotentia associated with abnormal slow activity constitutes evidence of a hypofunctioning cortex or of one which is spatially relatively remote from the recording electrodes. The diagnoses suggested by such findings, listed in the order of their probability, are: degenerative changes (including atrophy, gliosis, etc.), subdural hematoma, vascular and metastatic tumors. A consistent hypopotentia without abnormal activity is still suggestive of such changes.

4. A consistent hyperpotentia associated with abnormal slow activity is strong evidence of an irritative focus ordinarily to be associated with either a brain tumor or a Jacksonian convulsive state. The latter diagnosis is particularly suggested when such an irritative focus is associated with either a focal or diffuse dysrhythmia of a paroxysmal character. A consistent hyperpotentia without abnormal slow activity is in effect the same as a hypopotentia of the homologous areas under consideration. Although compatible with an irritative focus in the regions of the hyperpotentia, it is ordinarily to be interpreted in terms of a focal hypopotentia of the opposite side as explained above (see 3).

5. A consistent area of relative hypofrequency and of irregular or altered wave form suggests cortical changes similar to those associated with a focal hypopotentia without abnormal slow activity (see 3 above).

6. An area of hypopotentia associated with abnormal slow waves occasionally may be transformed into an irritative focus (see 4 above) or again a consistent irritative focus may be found in or adjacent to an area of hypopotentia. These combinations have been associated with degenerative processes and frequently with Jacksonian convulsive states.

7. A consistent hypopotentia in the regular recording has occasionally

shifted to a hyperpotentia under the influence of hyperventilation and has been interpreted as a potential irritative focus associated with a degenerative process.

These criteria of evaluation, which have been developed empirically in the course of this study, must be regarded as tentative only and subject to changes as may be dictated by further experience.

RESULTS

The group of 1200 cases studied comprised 393 convulsive states of unknown etiology, 238 convulsive states of the secondary type, 85 brain tumor

TABLE 1

Electroencephalographic classification of 1200 records obtained by a quantitative and statistical analysis of homologous tracings and correlated with the results of other diagnostic methods

E.E.G. Classification	Clinical		Pneumo-encephalography		Pathology	
	No. of Cases	% Correl.	No. of Cases	% Correl.	No. of Cases	% Correl.
Normal	172	91	15	100	3	100
Generalized dysrhythmia	450	90	81	84	8	50*
Total non-focal	622	90	96	87	11	64
Total focal	404	84	152	77	74	100
Slight	208	90	77	78	26	100
Gross	196	80†	75	76	48	100
Total correlations	1026	88	248	81	85	95
Borderline unclassified	174	—	—	—	—	—
Grand total	1200	—	—	—	—	—

* The 4 cases with pathology that failed to correlate and showed a generalized dysrhythmia were subcortical tumors of the 3rd and 4th ventricles.

† See text for explanation of the relatively low percentage correlation of the gross focal dysrhythmias with clinical findings.

suspects, 107 cases of degenerative diseases of the central nervous system, 147 post-traumatic head conditions, 123 psychiatric states and 107 miscellaneous conditions.

The electroencephalographic findings and their degree of correlation with the results obtained by other diagnostic methods are presented in Table 1. The data clearly indicate that evidence of focal cortical pathology is infrequently demonstrated by other methods when such evidence is lacking electroencephalographically. Conversely, when evidence of focal pathology has been found by this technique of electroencephalography, it may be cor-

related with similar data obtained by other diagnostic methods in a high percentage of cases.

The value of the proposed technique, however, does not lie in the high degree of correlation achieved in normal or grossly abnormal groups. In these respects, the results obtained with this technique correspond well with the results obtained by other electroencephalographic techniques. The value of the proposed method lies in its localizing ability in a very significant intermediate group in which the dysrhythmias were so slight that they could not be detected with any degree of certainty by techniques that fail to employ the principle of a statistical analysis of adequate data obtained by the simultaneous recording of homologous potentials. With the object of properly evaluating the proposed method, therefore, we have critically reexamined[1] our entire data and have divided the focal dysrhythmias into "Slight" and "Gross" subclassifications. On this basis, it is estimated that 20 per cent of the entire group which could be definitely classified electroencephalographically (208 cases out of 1026) were "Slight" focal dysrhythmias that probably could not have been detected by other techniques.

The difference in the degree of correlation between the clinical findings and the two subclassifications of the focal dysrhythmias may in large part be explained by the differences in the data utilized in arriving at these electroencephalographic diagnoses. The "Slight" dysrhythmias* were identified by relatively slight differences of potential, form, frequency and synchronism in homologous recordings obtained simultaneously as explained previously. The significance of such relatively slight changes was established by the statistical analysis of numerous recordings. Obvious focal delta activity, on the other hand, was classified as "Gross" and included numerous cases of convulsive states with shifting foci not necessarily associated with focal pathology or localized findings by clinical study. It is of interest in this connection that the electroencephalographic changes found by the statistical method of comparing homologous areas are constantly reflected in the record and that this type of data correlated better with the clinical findings than many of the more obvious but sometimes ephemeral dysrhythmias of focal character.

Because of the differences in the types of data that distinguish the "Slight" and "Gross" focal dysrhythmias, these subclassifications represent fairly definite groups, which can be determined with relative ease and certainty. This fact, in addition to the established correlative value of the "Slight" focal dysrhythmias, would appear to confirm beyond question the value of the method.

A good correlation with the electroencephalogram showing focal dysrhythmias was found in 77 per cent of 152 patients subjected to pneumoencephalography or ventriculography. It should be emphasized that this result included several deep lesions, the diagnosis of which could not reasonably

* Since most "Slight" dysrhythmias are not true dysrhythmias at all, in the usual sense of this term, it may be less confusing to use the term "focal changes."

have been expected by any electroencephalographic technique. The overall correlation between electroencephalography and pneumoencephalography, including ventriculography, in 248 patients was 81 per cent.

The 100 per cent correlation of focal dysrhythmias with pathology as established by operative procedure or autopsy is undoubtedly fortuitous in spite of the relatively large number of cases involved. This is indicated by the poor results obtained in the group showing a generalized dysrhythmia, which happened to include 4 deep lesions, whose discovery could not have been expected by electroencephalographic methods.

In evaluating the degree of correlation obtained between the electroencephalographic results and other diagnostic techniques, an analysis of those cases that failed to show focal changes is as valuable as the data concerned with focal dysrhythmias. Of 622 examinations in this group that failed to show any electroencephalographic suggestions of focal changes, clinical signs or symptoms referable to a cortical localization were observed in only 10 per cent. Pneumoencephalography was performed on 96 of these patients and revealed definite cortical pathology in 12 instances. Pathological information was obtained in only 11 of this same group, of which four showed focal pathology that had been missed by the described technique of electroencephalography. As mentioned above these were deep midline tumors of the 3rd and 4th ventricles and were associated with generalized dysrhythmias.

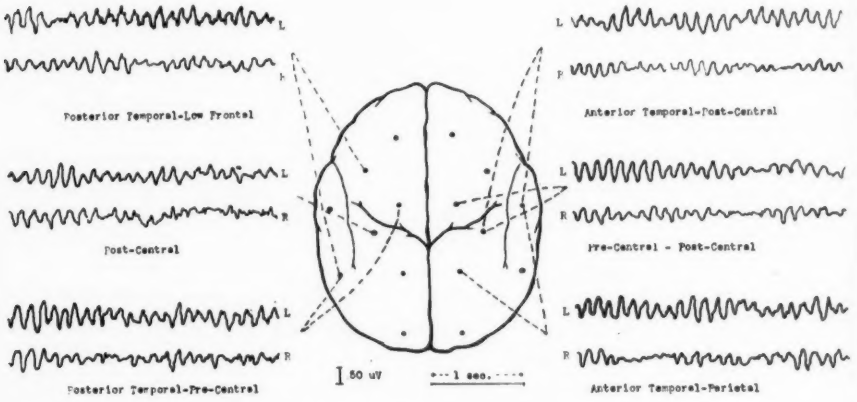
DISCUSSION

That the additional data obtained by the method of the simultaneous recording of homologous potentials are of value for purposes of localization may be illustrated by the following example.

H. K., male, 49 years, had experienced right-sided Jacksonian spells of progressive severity and frequency over a period of 5 weeks. Minor episodes, limited to the right hand and forearm, had occurred once or twice a day and lasted from 1–2 minutes. There had been four or five severe attacks involving the entire right side or both sides and associated with partial or complete loss of consciousness. The past and family histories were non-contributory. Positive neurological findings were limited to an inequality of the pupils, the right being larger than the left, deviation of the tongue to the right and a right spastic hemiparesis of slight degree. At the time of the initial electroencephalographic test on this patient (Figs. 4 and 5), the pattern was not disturbed by activity of abnormal form or frequency and localization by other techniques would have been impossible. The diagnosis was made solely upon the basis of asynchronous hemispheric discharge, inequalities of homologous potentials and relative differences in the form and frequency ranges of bilateral tracings recorded simultaneously. Approximately 7 months later another electroencephalogram was made. The summary analysis or "Spot Graph" (Fig. 6) showed a "Gross" focal dysrhythmia in the left temporoparietal region with a marked accentuation of the findings obtained in the initial study. This corresponded well with the increase in the right spastic hemiparesis exhibited by the patient at the time of the second electroencephalogram. Pneumoencephalograms obtained at the time of the second electroencephalogram were inconclusive and a left frontoparietal exploratory craniotomy, performed shortly after the second study, failed to reveal the cause of this patient's difficulties. An autopsy obtained 3 months later showed a meningeal fibroblastoma of the left parietal region.

As a result of repeated experiences of this type, it is our feeling that a review of the record, with attention focused on the region of suspected abnormality and with data emphasizing bilateral hemispheric differences, affords the best possible opportunity of detecting focal changes.

The value of the "Spot Graph" in ruling out stray variants that may be disregarded and focusing attention on the abnormal region is worthy of em-



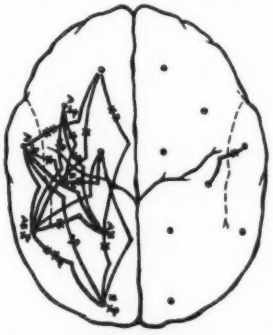
SIMULTANEOUS RECORDING OF HOMOLOGOUS AREAS

M.K. ♂ 49: 4/6/42 Right Jacksonian seizures of 5 weeks duration.
Examination: Right pupil larger than left. Tongue deviates to right. Right deep reflexes slightly hyperactive. Right leg weaker than left.
Laboratory findings: Negative. CSF pressure - 125 mm H_2O.
EEG. Suggestive focal changes in the left temporal and parietal regions compatible with an irritative focus and possible associated pathology.
Pneumoencephalogram: Generalized increase in subarachnoid spaces - most marked in the right temporal region.
Diagnosis: Right Jacksonian convulsive state. ? Brain tumor.

FIG. 5. Simultaneous recording of homologous areas.

phasis. The importance of the "Spot Graph" lies in the fact that it is a simplified statistical method of checking the results and placing electroencephalography on an objective basis. By "statistical" we refer to the fact that the proposed method (16-electrode technique as used for localization) involves approximately 10 recordings with respect to each electrode and that more than one or two abnormal runs, which involve the same electrode, are required to raise a suggestion of abnormality in the underlying cortex. A high percentage of records will show occasional abnormal runs, which have been termed "stray variants" and which, with the use of the statistical approach proposed, may safely be dismissed as without significance. Were these obtained in a routine involving fewer runs of longer duration, however, accepted on their face value and read into the record, the resulting interpretation would be very different. Electroencephalography is at best a difficult technique and even with the greatest care inconsistent or "variant" tracings appear. Our results amply confirm the soundness of the proposed quantitative and statistical method.

The degree of correlation of electroencephalographic localization in patients with proved lesions is essentially the same in this study as that obtained by various workers using other techniques.[3,4,5] It should be noted, however, that the focal dysrhythmias usually associated with such lesions are sufficiently gross to ensure a localization regardless of the technique employed. In this group the information obtained by the method of homologous recordings is of confirmatory value only. In addition to this group,



Legend

> Unequal potentials - left greater than right
= Equal monopolar potentials
Sp Poor synchronism of homologous potentials
Xp Very poor synchronism of homologous potentials
X Asynchronism of homologous potentials

E. K. ♂ 49: 10/29/42 Right Jacksonian seizures of 7 months duration.
Examination: Right spastic hemiparesis with increased deep reflexes and diminished superficial reflexes. Astereognosis and impairment of two point discrimination on right.
Laboratory findings: Negative.
EEG: Focal dysrhythmia centering in left tempore-parietal region compatible with a right sided Jacksonian convulsive state and pathology (?brain tumor).
Pneumoencephalogram: As before, except size of subarachnoid spaces less than on previous examination.
Operation: Negative exploratory craniotomy.
Autopsy: Meningeal fibroblastoma - left fronto-tempore-parietal region.

FIG. 6. Head diagram—"Spot Graph." H. K. 10-29-42.

however, definite localizations have been made in the present study in over 200 cases or approximately 20 per cent of the entire group, which could be definitely classified electroencephalographically. A high percentage of these diagnoses could not have been made by other techniques which do not employ the principle of a statistical analysis of homologous recordings. These localizations correlated well with the clinical findings (90 per cent correlation) as well as with the findings by pneumoencephalography in 77 cases of this group (see Table 1). A good correlation between the electroencephalographic, pneumoencephalographic and clinical findings cannot be discounted.

The extra time and effort required by the technique of homologous recordings are not excessive, as indicated by the routine use of this method in the 1200 examinations that form the basis of the present study. It is axiomatic, in electroencephalography as in other matters, that one finds only that for which one looks. The technique must be adequate for the task at

hand and for purposes of localization must employ a sufficient number of electrodes and runs to cover the available cortical areas, to rule out artifact and to establish the diagnosis on an objective basis. The quantitative and statistical approach proposed represents an effort in this direction and is reported in the belief that if electroencephalography is to be used for purposes of localization, it should be done thoroughly and well merits the slight extra time and trouble required.

CONCLUSIONS

1. A modified electroencephalographic technique based upon the quantitative and statistical analysis of homologous recordings obtained simultaneously has been investigated for its cortical localizing value. The principle involved is identical with that employed in the neurological examination in which focal abnormal findings on one side are checked against the normal side as a control. The technique, methods of analysis and principles of evaluation are presented in detail.

2. This technique has been employed in 1200 examinations on patients who have received an adequate clinical work-up. Definite cortical localizations were obtained in approximately one third of this abnormal group, which gave a high degree of correlation with the clinical findings. Ninety per cent of the remainder had convulsive states or other conditions in which focal signs and symptoms or neurological findings of localizing value were not obtained.

3. In 248 patients of this group subjected to pneumoencephalography, an over-all correlation of 81 per cent was observed between the roentgenographic findings and those of the electroencephalogram obtained by means of the technique of homologous recordings.

4. In 85 proved cortical lesions an electroencephalographic correlation of 95 per cent was obtained.

5. It is estimated that electroencephalographic localization of lesions of a less gross character was made in approximately 20 per cent of the entire group, which correlated well with the clinical and pneumoencephalographic findings and which would not have been detected by other techniques.

6. The extra time and effort required by the technique are not excessive and appear to be well justified when used for purposes of localization.

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ANTERIOR RHIZOTOMY

THE ACCURATE IDENTIFICATION OF MOTOR ROOTS AT THE LOWER END OF THE SPINAL CORD

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At the meeting of the American Society of Neurological Surgeons held in Montreal on October 20, 1944, Dr. Donald Munro[2] stimulated the members by a presentation on "Care of the Paraplegic." He showed movies of a number of patients completely paralyzed who were able to walk with the help of braces and crutches. The morale of these patients appeared to be excellent, and their general physical condition good. A number of them were earning their own livelihood. At this meeting, he stated that he had been unable to deal adequately with a certain group of patients who had marked spasticity and mass reflexes. These patients would not tolerate splinting. In a few of these he had converted the spastic paraplegia into a flaccid one, by cutting the anterior motor roots from thoracic ten to sacral one inclusive, and thus retaining the innervation of the bladder. This operation would seem to have a very definite place in the treatment of a certain group of cases and is a surgical procedure that will likely be carried out in many neurological centres.

Experience with one case by one of us (K. G. McK.) demonstrated the difficulty of identification of the roots. The surgeon must have accurate anatomical knowledge and a definite plan to identify the root, otherwise the operation can be very confusing.

Elsberg[1] stated that "the dentate ligament ends below, at the level of the first lumbar vertebra, in a fork-shaped extremity. . . . The first lumbar posterior root rests upon this fork, so that the 'fork' may be used as an anatomical landmark for the identification of the first lumbar root." He recommended that other roots should be recognized by counting in either direction from this first lumbar sensory root.

Anatomists agree that the highest process of the denticulate ligament is attached to the margin of the foramen magnum. There is disagreement, however, on the number of denticulations throughout the length of the ligament. A table of various opinions follows:

Number of ligamentous processes	*Authority*
21	Parsons. *Gray's Anatomy.* 26th ed.
About 21	Piersol. *Human Anatomy.* 9th ed.
20 to 22	Cunningham. *Text-book of Anatomy.* 8th ed.
20	A. Lee McGregor. *A Synopsis of Surgical Anatomy.* 4th ed.
19 to 23	Emil Villiger. *Brain and Spinal Cord.*
21	Tilney and Riley. *The Form and Functions of the Central Nervous System.*
About 21	Morris. *Human Anatomy.* 10th ed.

Usually 21	Werner Spalteholz. *Hand Atlas of Human Anatomy.* Vol. 3.
20 to 25	Sobotta and McMurrich. *Atlas and Text-book of Human Anatomy.* Vol. 2.
About 21	Quain's *Elements of Anatomy.* 11th ed., vol. 3, part 1.

It is apparent from these observations that the lowest attachment of the denticulate ligament is at a variable vertebral level, and thus has not a constant relation to the first lumbar posterior root. From dissections of 10 cadavers, it is concluded that this anatomic landmark is inconstant. A table of results follows:

Subject	*Root lying on lowest slip of the denticulate ligament*	
	Left	*Right*
A	L-2	L-2
B	L-1	L-1
C	L-1	L-2
D	L-1	L-1
E	L-1	L-1
F	L-2	T-12
G	L-2	L-1
H	L-2	T-12
I	L-2	L-2
J	L-1	L-1

In 4 cases only, of the 10 cadavers dissected, were the posterior roots of the first lumbar nerves lying on the lowest slip of the denticulate ligament on both sides of the cadaver. It is important to note that in 2 cases, the twelfth thoracic root on the one side and the second lumbar root on the other side would have been identified as first lumbar roots by Elsberg's method of identification.

Early in the dissections, it was apparent that counting the roots at the lower end of the spinal cord was extremely difficult and inaccurate, due to their close approximation to each other.

It is important to realize, therefore, that if an upper lumbar root is definitely identified, the inaccurate procedure of counting other lower roots would still be necessary.

THE ACCURATE IDENTIFICATION OF ROOTS AT THE LOWER END OF THE SPINAL CORD

In an effort to eliminate the uncertain process of counting roots and because the lowest tooth of the denticulate ligament was not an accurate guide, a method was sought of identifying the lowest possible root of those to be divided.

Observations on an additional 4 cadavers and a study of 2 cases at autopsy indicated that the first sacral motor root could be identified accurately because it is the lowest large root to leave the conus medullaris. The difference in size between the large first sacral root and the much smaller second sacral root is so striking that there is no special difficulty in distinguishing between them. In this second series of 6 dissections the first sacral root was identified at the lower end of the spinal cord by its size and position. The identification was found to be correct, bilaterally, in each case by following

the root to its exit through the first sacral foramen. It is essential in making this identification to obtain a satisfactory view of the lower end of the cord. In actual operation it has been done in two ways:

(a) A loose ligature is passed around the filum terminale avoiding trauma to the slender, friable, coccygeal roots. A blunt probe is passed downwards along the plane of the denticulate ligament and all the lower sensory roots on that side are hooked up *en masse* and held medially. The loose ligature is grasped and the filum terminale is pulled in a cranio-lateral direction between the bundles of anterior and posterior roots, into a position lateral to the medially retracted dorsal roots.

The spinal cord is rotated to bring the anterior roots clearly into view by traction on the lowest process of the denticulate ligament and the filum terminale. Two or three contiguous roots may hinder this manoeuvre and can be cut without any deleterious results. The lowest big anterior root may then be definitely identified as the first sacral and cut, and all motor roots above sectioned up to the desired level. The procedure is repeated on the opposite side. (See Fig. 1.)

(b) A second, and probably more simple method may be carried out by severing the lowest tooth of the denticulate ligament and retracting the posterior roots as above. If a few of these sensory roots, which are too taut for adequate retraction, are cut, a good view of the motor roots may be obtained.

The main advantage of this latter procedure is that the laminectomy does not have to be carried down quite so far, as the filum terminale does not have to be visualized as in (a).

The only disadvantage of (b) is that the view of the lower roots is not as extensive as in (a) where the motor roots are put on the stretch which gives a clearer picture of the anatomy of this region.

The identification of the highest root to be cut presents some difficulty; although accurate identification here is not as important as identification below where the supply of the bladder must be considered. It is suggested that accurate identification at this upper level should be made by placing a silver clip on what is judged to be a lower thoracic motor root at its exit from the dural canal. A portable x-ray plate will then accurately identify this clipped root in relation to the ribs and vertebrae. A pre-operative x-ray film to rule out vertebral or costal anomalies would be an additional precaution to insure accuracy. The necessary number of roots to be cut in the upper part of the exposure can then be counted, as the roots in this region are spaced sufficiently far apart to render this procedure easy.

To gain adequate exposure for the sectioning of the tenth thoracic root to the first sacral, it is necessary to remove the spines and laminae from the tenth thoracic vertebra to the second lumbar vertebra.

Through the cooperation of Professor J.C.B. Grant of the Department of Anatomy in the University of Toronto, one of us (I.B.M.), while a Demonstrator in Anatomy, was given the privilege of carrying out the series of dissections.

CLINICAL NOTES

In this centre, anterior rhizotomy has been performed on 6 patients because mass reflexes prevented adequate nursing care, the prevention of bedsores and efficient care of the genito-urinary tract. In all of these 6 patients,

mass reflexes have been abolished. In 5 instances identification of nerve roots was done by the method suggested above.

In contrast to Munro's experience, permanent disturbance of bladder function has followed the operation in 2 patients. Pre-operatively, these pa-

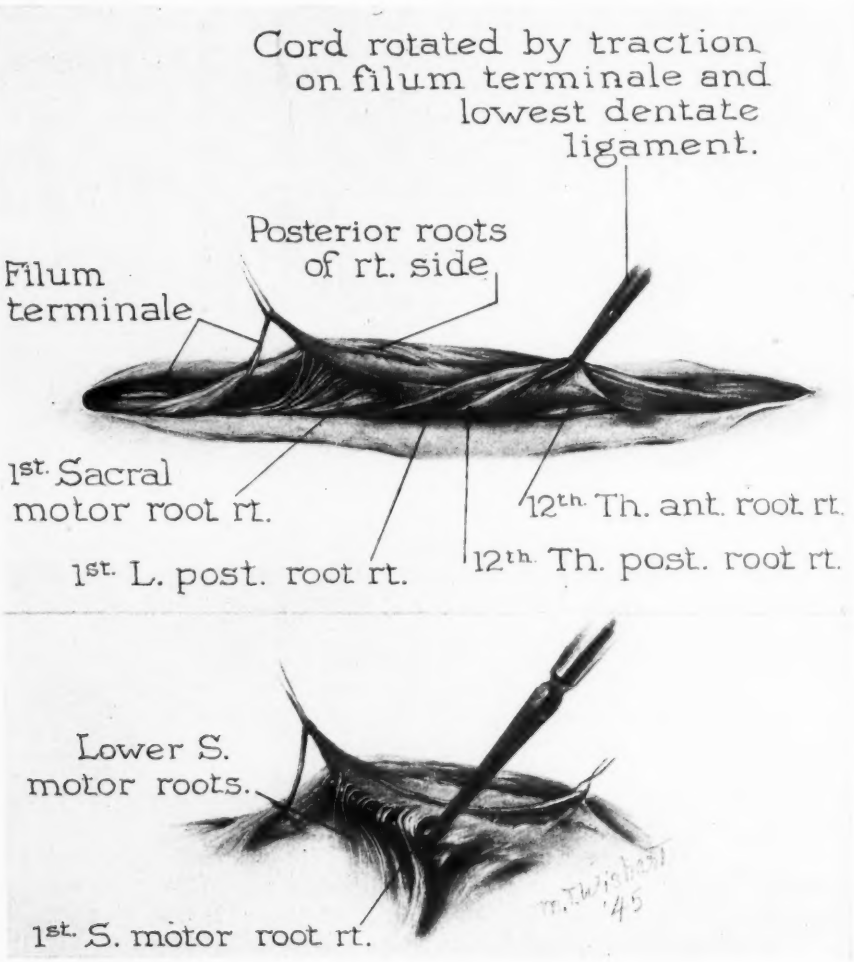
Cord rotated by traction on filum terminale and lowest dentate ligament.
Posterior roots of rt. side
Filum terminale
1st. Sacral motor root rt.
1st. L. post. root rt.
12th. Th. ant. root rt.
12th. Th. post. root rt.
Lower S. motor roots.
1st. S. motor root rt.

FIG. 1.

tients had bladders capable of good reflex contractions. Following operation, the bladder was completely flaccid and has remained so for three months or longer. Of the remaining 3 patients, reflex activity of the bladder was unchanged in two and in one patient cutaneous ureterostomies prevented assessment of bladder function after operation.

At the moment, from this experience, it is our belief that:

(a) There is the possibility, although in our opinion unlikely, that the roots were inadequately identified at operation.

(b) In certain patients, the first sacral root may be necessary for adequate reflex activity of the bladder.

(c) We believe that the most likely explanation for the abolishment of reflex activity of the bladder in these two, possibly three, patients is as follows: Section of a large number of anterior roots may cut off sufficient blood supply to the conus medullaris to bring about loss of function in the sacral segments. At operation, it may be possible to save some of the arteries accompanying the anterior roots and every effort should be made to do so.

(d) One of us (E.H.B.) has concluded that in future he will leave the first sacral root, for persisting spasticity about the ankle joints will unlikely interfere to a significant degree with the retraining program with braces and crutches.

SUMMARY

1. The relation of the first posterior lumbar root to the lowest slip of the denticulate ligament is inconstant.

2. A method of identifying the first sacral motor root is suggested.

3. It is advisable to preserve as many anterior root arteries as possible when performing an anterior rhizotomy.

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HYDROCEPHALUS

A CONTRIBUTION RELATED TO TREATMENT

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THE FOLLOWING paper was read before the American Neurological Association in 1942 and appears in abstract form in the transactions of that year. I decided not to publish the original paper, however, until further clinical tests could be made. In pursuing the problem of congenital hydrocephalus I have arrived at conclusions that no doubt many others have deduced. It is only in the occasional and well selected infant having an otherwise reasonably normal brain that one can hope to have any gratifying success regardless of the method used. Successes in the treatment of congenital hydrocephalus are few and far between. This paper is presented, however, in its original form since it is felt that the reasoning is sound and that there is no other method of treatment at present that is superior to the one herein described. Moreover, my interest has been considerably augmented by the result in treatment of a child who acquired severe communicating hydrocephalus as a sequel of pneumococcic meningitis. The case and discussion are presented as an addendum to the original paper.

Hydrocephalus† has been a challenging and almost insuperable problem to the neurological surgeon. With a clear understanding of the origin, circulation and absorption of cerebrospinal fluid it has seemed incredible that a more satisfactory method of treatment has not been devised. The obstacles, however, have proved to be great. There have been two general methods of surgical treatment. (1) Some procedure designed to establish an absorbing mechanism for the cerebrospinal fluid. Every conceivable idea has been put

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† A clarification of terminology appears to be advisable if only to avoid a misinterpretation of my own statements. The term hydrocephalus means literally "water head" and has at times been used loosely to designate any case of dilated ventricles. I would prefer to restrict the usage of the term to only those cases in which a dilatation of the ventricles results from an obstruction to the circulation and absorption of the cerebrospinal fluid—back pressure. Dilated ventricles that result from congenital malformations of the ventricles themselves or from atrophy of brain substance without back pressure should be designated only as dilated or enlarged ventricles in order to imply a distinction relative to the causal factor. Back-pressure hydrocephalus might be designated *obstructive hydrocephalus* and would be correct, but I believe it would imply to many a ventricular-aqueductal obstruction. All back-pressure hydrocephalus is obstructive and the obstruction may be anywhere from the place of formation of the ventricular fluid to its place of absorption. The terms *communicative* and *non-communicative*, because of their common usage, would suffice to designate respectively whether the obstruction is distal or proximal to the foramen of Magendie, or more specifically the cerebellar cistern. Acquired and congenital hydrocephalus are sufficiently distinguishing terms. The former is overwhelmingly the result of neoplasia while the latter is seldom so. Yet hydrocephalus may be acquired in adult life as a result of adhesive arachnitis and hence present much the same surgical problem as does congenital hydrocephalus. When using the term hydrocephalus I intend to imply the type caused by lesions other than expanding masses unless otherwise specified.

to test. To mention a few—silk threads or tubes have been extended from the ventricles to the tissues of the scalp or to the muscles of the neck; tubes have been extended from the dural cul-de-sac into the peritoneal cavity (for communicating hydrocephalus). Cushing originated this idea and tried it in six cases. The patients succumbed and because they appeared to have intussusceptions at autopsy, Cushing (erroneously) thought the spinal fluid might contain too great a concentration of pituitrin to make the procedure feasible. I placed a silver tube from the dural cul-de-sac to the peritoneal cavity in an infant, and it served its purpose well but the infant later expired with hyperthermia.

The ideal objective would be the establishment of an artificial communication from ventricle to some available subarachnoid space of the brain. This has chiefly taken the form of an artificial opening in the floor of the third ventricle with the hope of making a permanent communication from that ventricle to one of the basilar cisterns. This method would be entirely futile in the event of an adhesive basilar arachnitis. There have been some successes,[1] more so in adults,[8] but on the whole, and particularly in infants, the method is unsuccessful because, as is true of all such methods where openings are made from the outside into a ventricle, the integrity of the arachnoid is disrupted and the ventricular fluid is not forced into the subarachnoid space but into the subdural space. This is one reason that openings into the ventricles through the corpus callosum were failures. The method would no doubt be more successful if the opening was made from a ventricle into the subarachnoid space (from inside out) while preserving the parietal arachnoid and assuming that such an artificial opening would not later close.

(2) In any case, the failures led surgeons to another type of treatment* designed to diminish or abolish the production of ventricular fluid by destruction of choroid plexus. The plexuses in the lateral ventricles are the only ones that lend themselves to a reasonably feasible attack. If they are completely destroyed, a considerable amount of plexus still remains in the third and fourth ventricles. Nevertheless, this method has in late years proved to be the most successful yet. Dandy[1] in 1918 removed the plexuses but it was done through an air cystoscope which necessitated removal of the ventricular fluid for a time and though the fluid was quickly replaced, the mortality must have been high especially when the ventricles were so large that the brain tended to collapse. I used the method in five cases, and no matter how quickly the ventricular fluid was again replaced they were all fatal. On the other hand, if the ventricular fluid is not withdrawn, the procedure is seldom fatal. This requires a lens system however. The lens system used in conjunction with bipolar electrodes with which the plexus is cauterized appears to be the most successful method yet devised in terms of results.[3,4,5,6,7]

* All more conservative methods to date, such as dehydration, are admissions of defeat and could not possibly be maintained. Decompressions and repeated spinal or ventricular punctures when intended as definitive treatment are obviously not physiologically sound and should be discouraged.

I have always been skeptical, however, that the good results were due only to a diminished production of fluid, because the total choroid plexus is by no means destroyed. Six years ago I cauterized a lateral plexus in an infant 4 months of age with advanced hydrocephalus. I used a unipolar elec-

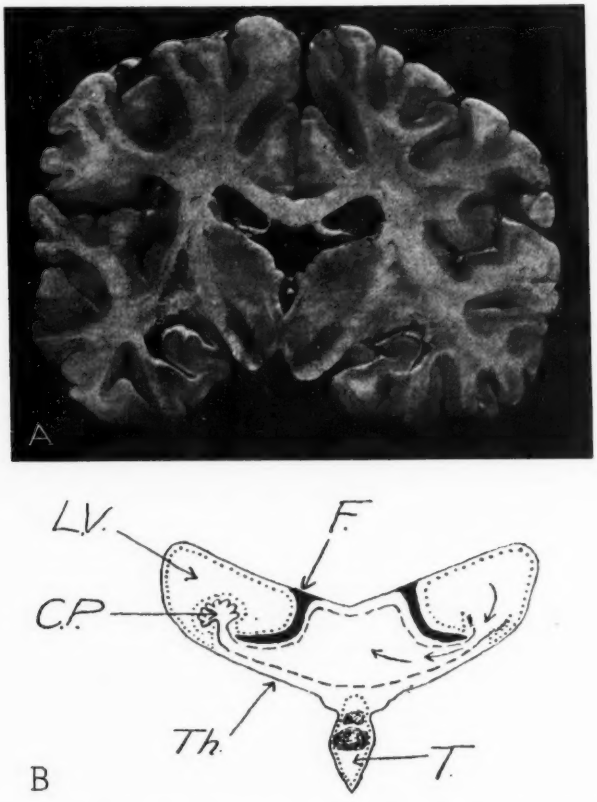

FIG. 1. (A) Photograph of coronal section of a normal brain, just anterior to the posterior commissure. The reader looks toward the rear of the brain. (B) Diagrammatic representation of the ventricular structures and the tela choroidea. Dotted line: ependyma. Broken line: pia-arachnoid of the tela. On the right the choroid plexus has been disrupted (shown only in the diagram). As shown by the arrows, ventricular fluid now flows from ventricle into subarachnoid space of tela (cisterna venae parvae). See Fig. 2 for lateral view. C.P., choroid plexus; L.V., left ventricle; F., fornix; T., posterior aspect of 3rd ventricle; Th., upper margin of thalamus.

trode, which is not very effective under ventricular fluid, but since I grasped the plexus with an alligator-jaw mechanism, I mutilated and tore it. The fontanelles closed and the increased intracranial pressure subsided and has remained so to the time of writing. I was always at a loss to explain the result because I knew I had destroyed relatively little plexus.

By applying an anatomical fact, I believe I can explain the good results that are obtained by mutilation or cauterization of the plexus on another basis than that of diminished formation of fluid. I propose to show that a disruption of the laminae of the choroid plexus establishes a communication

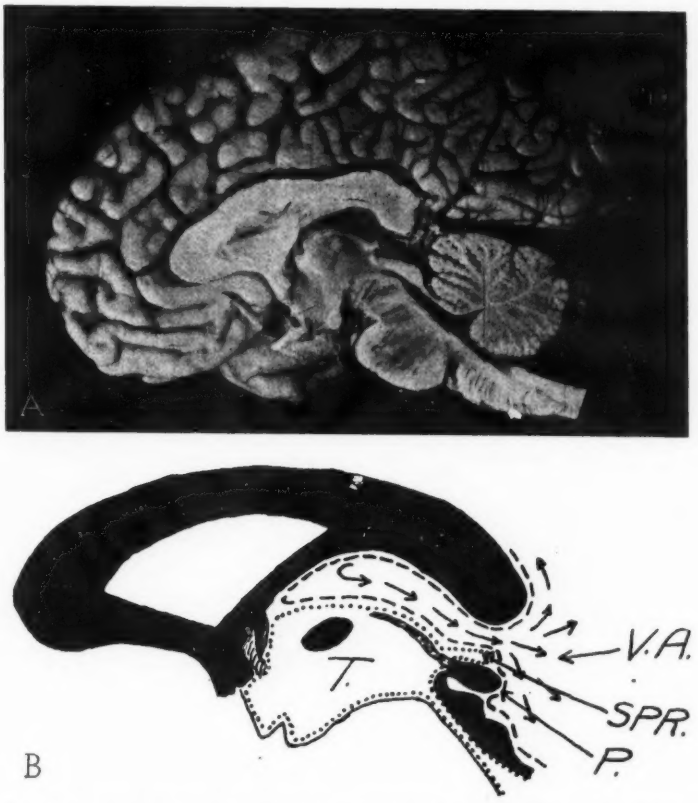

FIG. 2. (A) Photograph of mesial surface of a normal brain showing the extensive space occupied by tela and cisterns of the small and large veins of Galen and cisterna ambiens. The spaces are sponge-like in character due to their reticulated structure. (B) Diagrammatic representation of the tela and its surrounding structures. Dotted line: ependyma. Broken line: pia-arachnoid, the visceral arachnoid membrane. The parietal arachnoid membrane is not shown. The arrows show the course of ventricular fluid after it gains access to the spaces of the tela. It flows into the cisterna venae parvae, thence into the cisternae venae magnae and ambiens, and thence to all available subarachnoid spaces. T., 3rd ventricle; V.A., cisternae venae magnae and ambiens; SPR., suprapineal recess; P., pineal body.

from the lateral ventricle through the tela choroidea to the cisterna venae magnae and cisterna ambiens and thence to all available subarachnoid spaces of the brain. I believe this communication which has been unwittingly made by destruction of the plexus is the factor chiefly responsible for the successes being obtained. This being true, I also propose that the plexus might be

attacked on one side with this end in view and that the treatment is applicable to congenital hydrocephalus, communicating and non-communicating, of whatever cause, as well as being applicable to acquired hydrocephalus.

THE TELA CHOROIDEA

The tela choroidea (velum interpositum) consists of a fold of pia mater between the fornices and the ependymal roof of the third ventricle. For

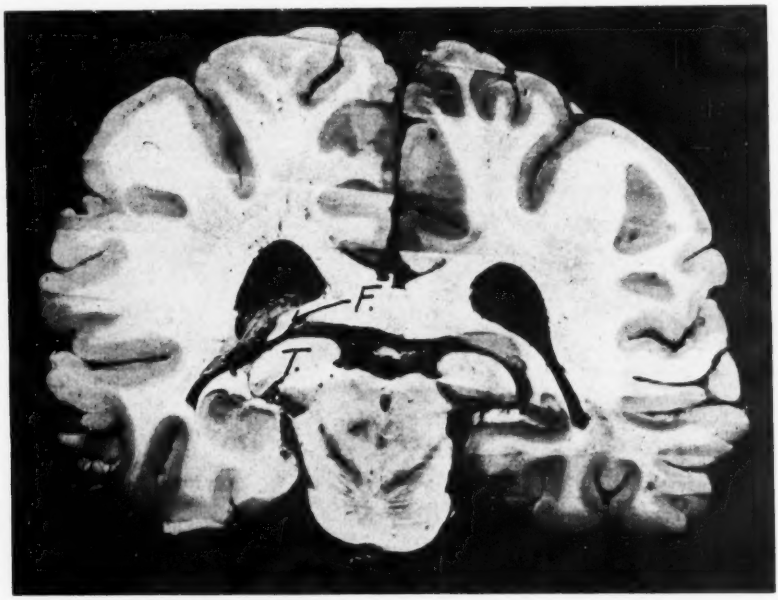

FIG. 3. Photograph of coronal section of a normal brain showing on the left the space between the fornix (F.) and the thalamus (T.). This is the choroidal fissure and is the space through which fluid may flow from the lateral ventricle into the cisterns of the small and great veins of Galen when the choroid plexus is disrupted.

convenience it may be visualized as the finger of a glove which in the developmental stages progresses forward between the fornices and the roof of the third ventricle. Hence, the forward end is closed while the posterior end opens into the cisterna venae magnae and cisterna ambiens. Thus, the inside of the glove finger is subarachnoid space and for which I would like to suggest the name *cisterna venae parvae*. The portion of the tela in contact with the ependymal roof of the third ventricle pushes the ependyma ahead of it to form the paired choroid plexuses of the third ventricle. The lateral aspects of the tela project between the fornices and thalami into the lateral ventricles to form the choroid plexuses of the lateral ventricles. The posterior aspect of the tela broadens until its lateral aspects project above the fimbria

hippocampi and into the temporal horns, there forming the plexuses of the temporal horns. The plexuses of the third ventricle are continuous with those in the lateral ventricles through the foramina of Monro, and as the plexuses in the lateral ventricles swing around the thalami posteriorly, they become continuous with the plexuses in the temporal horns. If one visualizes the tela as a glove finger which is flattened and has a very broad base, then the space into which it fits is known as the transverse fissure of the brain.

The choroid plexus at any point is supported by two delicate pial laminae,

FIG. 4. Photograph of coronal section of the brain with which the indigo-carmine experiment was performed. The intact brain was laid on its right side and a wedge cut into the left ventricle as shown. After plugging the foramen of Monro, the dye was poured into the ventricle. For 10 minutes the dye did not escape from any opening. Then the choroid plexus was gently torn. The dye rapidly escaped between the fornix (F.) and thalamus (T.) into the tela and thence into the cisterna ambiens. No dye gained access to the 3rd ventricle (T.V.).

and if the continuity of these laminae is broken at any point, a communication is established between the ventricles and the subarachnoid space (Figs. 1, 2 and 3). Normally the tela, with its outpushings of choroid plexus, is a water-tight intact structure open only posteriorly where it communicates with the cisterna ambiens. The only way that ventricular fluid can and does gain access to the tela is by first circulating through the foramina of Luschka and Magendie and thence into the general subarachnoid space. (It is interesting that a vascular modification of a portion of the arachnoid pours fluid into the ventricles which is destined to be absorbed in the subarachnoid spaces.) By disrupting the choroid plexus at any point in the

lateral ventricle one should be able to establish a communication between the ventricle and the cisterna venae parvae and thence into the general subarachnoid spaces of the brain.

EXPERIMENT

A normal fixed brain was placed on its side and a wedge of tissue removed from the upper side so as to expose a lateral ventricle. The foramen of Monro was sealed with cotton and collodion. The lateral ventricle was then filled with indigo-carmine solution. After 10 minutes the dye was still in place. Then with forceps the choroid plexus was gently torn and immediately the dye poured out through the cisterna ambiens. The brain was sectioned coronally and

FIG. 5. Sagittal section of normal (formalin fixed) brain showing India-ink suspension in the tela choroidea. After the dura was cut away from the occipital lobe, ink was found to have migrated into its subarachnoid spaces (S.S.). This can be seen in the photograph. No ink gained access to the 3rd ventricle (T.V.).

the fact established that the dye went between the torn laminae of the plexus into the spaces of the tela and thence poured out through the cisterna venae magnae (Fig. 4).

Two other brains (after fixation in formalin) were treated similarly using dilute India ink instead of indigo-carmine as a dye. These brains had been removed at autopsy in such a way as to preserve the dura with the brain. After tearing away the glomus of a plexus on one side

the ink progressed into the space of the tela freely. In one brain, in spite of fixation and also in spite of a low head of pressure, the ink migrated throughout the subarachnoid spaces of the occipital lobes. The brains were first divided by an incision through the mid-sagittal plane (Fig. 5). Coronal sections were then made at various levels from the foramina of Monro to the cisterna venae magnae. Sections were cut from blocks that included tela, fornix and choroid plexus. They were counterstained with eosin. A study of these sections showed clearly that the ink progressed between the torn laminae of the plexus and into the space of the tela. On the intact side the ink found its way between the laminae of the plexus and even migrated

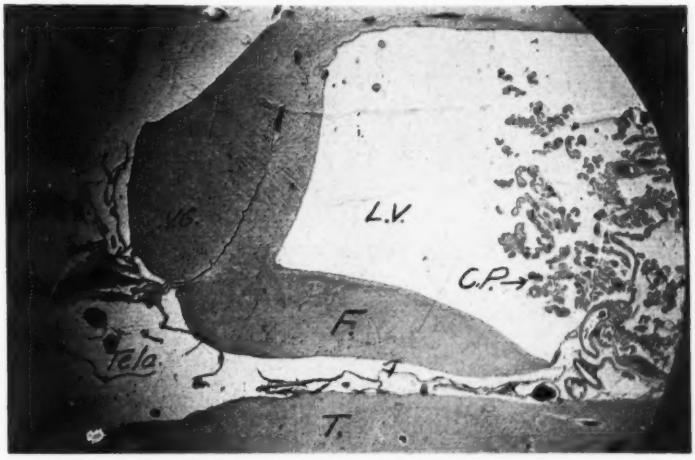

FIG. 6. Photomicrograph of coronal section through the tela choroidea of specimen shown in Fig. 5. The section shows a fornix and thalamus on only one side—the side opposite to that whose choroid plexus was torn. Ink had disposed itself throughout the space of the tela (cisterna venae parvae) and had progressed between the fimbria of the fornix and thalamus to some distance in the meshes of the intact choroid plexus. There was no evidence that ink had escaped from the plexus into its lateral ventricle. F., fimbria of fornix; T., thalamus; C.P., choroid plexus; L.V., lateral ventricle; V.G., a small vein of Galen.

into the connective-tissue meshes of the intact plexus on the opposite side, but there was no evidence that it escaped from the intact plexus into the ventricle on that side (Fig. 6). Sections from an occipital lobe showed that the ink had found its way into the subarachnoid spaces of the sulci and deep into the perivascular spaces (Fig. 7).

MANAGEMENT OF CONGENITAL HYDROCEPHALUS

Given an infant with advancing hydrocephalus obvious at birth or shortly thereafter, the chances are so great that the hydrocephalus is not due to a surgical lesion, such as tumor, one may reasonably disregard such a diagnosis. The usual cause will be atresia or absence of the aqueduct of Sylvius, Arnold Chiari malformation or adhesive arachnitis.* One may determine whether

* I have seen one case in which a non-patent sagittal sinus was thought to be the responsible factor for hydrocephalus. The circulatory pathways were all patent including well developed and somewhat deepened subarachnoid spaces over the cortex.

the hydrocephalus is communicating or non-communicating by injecting one or two cc. of phenolsulphonphthalein into a ventricle through the lateral aspect of the anterior fontanelle and attempting to recover it in the spinal fluid in one-half hour. Usually the ventricles are too large for ventriculography to be practicable.

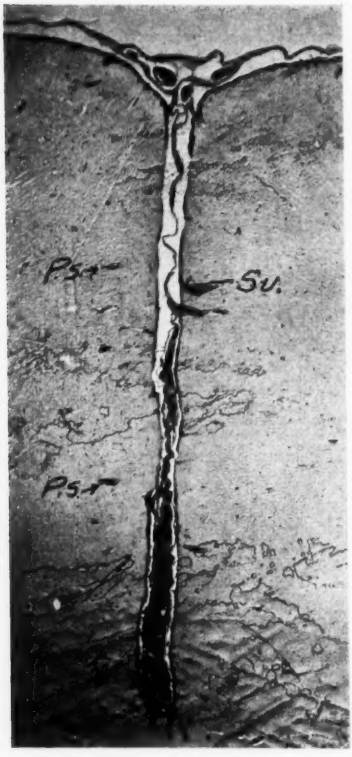

FIG. 7. Photomicrograph of section taken from the occipital cortex, roughly at the place marked S.S. in Fig. 5. Ink had progressed into the subarachnoid spaces of the sulci (Su.) and was prominent in perivascular spaces (P.S.).

If an Arnold Chiari malformation should be present, and it may be associated with a meningocele, it is probably wiser not to attempt a repair. Young infants do not tolerate such major operations well and even a removal of the herniated cerebellar tonsils does not guarantee a correction of the hydrocephalus. It is also probably wiser to attempt correction of hydrocephalus, if it is present, before repairing an associated meningocele. In short, without regard for the possible underlying cause of the hydrocephalus, the indication would be an attempt to correct the hydrocephalus by disrupting the choroid plexus on one side, thus establishing a communication from the ventricles to the cisternae venae magnae and ambiens. Even if the obstruction is due to a basilar arachnitis it is not necessarily likely that the upper subarachnoid spaces are involved and in any case if all the subarachnoid spaces are closed, the outlook is well nigh hopeless unless a destruction of the lateral plexuses can in itself effect an arrest of the hydrocephalus.

If the head is large and the calvarium delicate, it is well to encase it in a light cast as suggested by Dandy,[1] for the purpose of maneuvering it because when the intracranial pressure is suddenly relieved, the soft head is subject to damage. Openings are made in the well padded cast over the anterior fontanelle and over the parieto-occipital boss on one side. With a sharp needle the ventricle is punctured through the lateral angle of the anterior fontanelle and some fluid withdrawn.* This should be done

* It is most interesting and puzzling to note that fluid from such ventricles does not have the appearance nor the chemical analysis that characterizes stagnating cerebrospinal fluid outside of the ventricles. Putnam[6] states that the ventricular fluid in ordinary hydrocephalus rarely contains more than 15 mgm. per cent of protein. Seven years ago I had careful chemical studies made on the ventricular fluid of an infant with advanced hydrocephalus. Dr. B. E. Clark in the Department of Physiological Chemistry

preparatory to operation because infants often react to the mere release of long-standing pressure by a high fever—of even hyperthermic proportions. They will usually recover, however, but the puncture should be repeated daily or every two days until this particular type of reaction no longer occurs. I feel that it is quite advantageous to tide the baby over this particular reaction in the simplest possible way so that it will not complicate the effects of the more serious operation.

The operation may then be carried out by placing the baby on its abdomen with the parieto-occipital boss the uppermost part of the calvarium. The opening at this point need be no larger than a burr hole. The cortex is incised and a lens system introduced. Any means of tearing or disrupting the choroid plexus may be used. I have merely grasped the glomus with an alligator-jaw mechanism and pulled it away. No doubt an initial cauterization of the plexus would diminish bleeding and improve the procedure but bleeding has not appeared to be serious.

ACQUIRED HYDROCEPHALUS (NON-NEOPLASTIC)

It goes without saying that before making this the essential diagnosis in an adult every means should be used to rule out the presence of surgical pathology as the causal factor. I have seen only two cases in which a final diagnosis of basilar arachnitis could be made, excluding cases of clinically evident meningitis. The first patient died of progressive hydrocephalus before I had learned of the present method of treatment. The evidence of increasing intracranial pressure, and hence probably the adhesive arachnitis, began during the convalescence from pneumonia. The second is discussed as Case 4. Both of these at first had a communicating hydrocephalus. I have encountered three cases of non-communicating hydrocephalus in adults. They proved to have acquired a thin pial membrane over the foramen of Magendie, the rupture of which provided a permanent cure for the two who survived. Hence when the hydrocephalus is non-communicating and ventriculography shows the ventricular system to be dilated to the foramen of Magendie without other deformity, a cerebellar exploration is indicated.

If the diagnosis is finally a progressive hydrocephalus due to basilar arachnitis, the posterior horn of a lateral ventricle on one side may be opened through an incision in the brain and the choroid plexus cut away under direct vision and the usual hemostasis. In these cases removal of the ventricular fluid at the time of operation does no harm.

In the event of an inoperable posterior fossa tumor and the surgeon feels

reported figures on protein, sugar and chlorides that were well within normal limits. The infant died in the hospital 3 weeks after birth. India-ink suspension was injected into the ventricles before the brain was removed. The aqueduct of Sylvius was absent and the fact was established that there was no point of exit from the ventricles. Even if ventricular fluid is absorbed to some extent in and about the ependyma, the fluid in this case would certainly have to be regarded as stagnant. Such findings would strongly indicate that products of metabolism are not emptied into the ventricles and that the latter act as an inanimate container. If the cerebrospinal fluid serves as the lymph of the brain and cord and sweeps the products of metabolism from cell to blood, then this process must occur entirely outside the ventricles. The protein content of spinal fluid below a block very soon reaches an excessive figure.

it advisable and necessary to establish some means of cerebrospinal fluid circulation which might not have been improved by the cerebellar decompression, it would be an easy matter to make an incision into a posterior horn through a small craniotomy and remove the glomus of the choroid plexus. Aside from the fact that such a procedure is more physiological than the permanent insertion of a tube from ventricle to cerebellar cistern, it probably has another advantage. I have had reason to believe that cerebellar tumors obstruct the circulation of cerebrospinal fluid not only by obliterating the aqueduct and fourth ventricle but also by obstructing the cisterna pontis and cerebellar subarachnoid pathways in general. Furthermore, the cerebellar tonsils are usually squeezed into the cervical spinal canal in such a way as to trap fluid in the cerebellar cistern. Hence, even if a tube is placed from ventricle to cerebellar cistern, there is no guarantee that the fluid will reach its necessary destination.

It is well to inject phenolsulphonphthalein into the ventricle both before and after operation and look for its output in the urine at 15-minute intervals. In this way some evaluation can be made of what has been accomplished by the operation herein described because the rapidity and quantity of output in the urine is an indication of the quantity of absorption that has been occasioned in the subarachnoid spaces.

CASE REPORTS

FIG. 8. Case 1. An extreme hydrocephalus due to absence of the aqueduct of Sylvius.

Case 1. L.M., an infant 8 months of age, was admitted on Sept. 29, 1941, with an obvious hydrocephalus (Fig. 8). The greatest circumference of the head measured 72.5 cm. The parents stated that the head started enlarging at the age of 6 weeks. The ventricular pressure was 270 mm. of water in the prone position and the fluid was water clear; 200 cc. were removed. In 12 hours the temperature reached 104.2° F. and finally 105.6°. It then subsided precipitously. Fluid was withdrawn on two more occasions with a 2–3° response in fever. One cc. of phenolsulphonphthalein injected into a ventricle could not be recovered in the spinal fluid in ½ hour. The spinal fluid was sparse and contained 560 mgm. per cent of protein. A light cast was applied about the head.

Operation. On Oct. 6, 1941 the glomus of the choroid plexus on the right was grasped with an alligator-jaw mechanism and pulled away. A lens system was used and the operation was done without removing ventricular fluid. An appropriate opening was made in the cast, the baby was placed on its abdomen and the lens system inserted through a small opening made at the right occipito-parietal boss which was uppermost. The approach is therefore through the occipital lobe. The brain at this point was about ⅛ inch thick. The operation was carried out under local anesthesia.

Course. There was no reaction to the operation but on the 7th postoperative day a ventriculitis unfortunately developed and pneumococcus type 20 was recovered. Sulphapyridine was administered and as much as one gram of sulphanilamide on three occasions was injected into the ventricles. The baby recovered from the infection and was discharged 33 days following operation.

Periodic ventricular pressures were measured. The pressure gradually subsided to the neighborhood of 120 mm. at the time of discharge.

One month after discharge from the hospital the baby died and a postmortem examination was done by Dr. F. W. Mulsow of Cedar Rapids, Iowa. He reported that the aqueduct of Sylvius was not patent. The ventricles contained between 3 and 4 quarts of fluid. The choroidal fissure created by removal of the plexus was still patent but no further test was made. There was no evidence of meningitis and the ventricular fluid contained only 60 lymphocytes per c. mm.

Case 2. T.B., 4½ months of age, was admitted on Oct. 6, 1941. The baby had been born prematurely and weighed 4½ pounds at birth. In addition to the hydrocephalus (Fig. 9) there was a marked depression of the sternum, which made respiration somewhat laborious.

Ventricular puncture through the anterior fontanelle revealed a pressure of 250 mm. of water in the prone position. Spinal fluid could not be obtained and it was presumed that the hydrocephalus was non-communicating.

There was no untoward reaction to the release of intracranial pressure and on October 9th the glomus of the choroid plexus in the right lateral ventricle was pulled away. When the trephine was made over the occipital region no brain was encountered. Only pia arachnoid was present, indicating an absence of at least part of the occipital lobe.

During the postoperative stay in the hospital the fontanelle remained quite soft and at the time of discharge (10th postoperative day) the intraventricular pressure measured 90 mm. of water in the prone position. Fluid intake had been normal. The baby died 2 months after discharge, presumably of upper respiratory infection.

(These two cases were poor risks but they nevertheless responded well to the operation.)

FIG. 9. Case 2. Presumed to be a non-communicating hydrocephalus.

Case 3. S.C., 10 months of age, was admitted on Dec. 3, 1941, with the diagnosis of hydrocephalus and occipital encephalocele.

The anterior fontanelle measured 4×5 cm. and the posterior fontanelle 1.5×1 cm. On Mar. 8, 1941 (a previous admission) the greatest circumference of the head was 41.0 cm. On this admission it measured 50.5 cm. It was decided that the hydrocephalus should be treated first and the encephalocele repaired later.

Dec. 7: The ventricular pressure measured 140 mm. of water in the prone position. This was not as high as was expected. One cc. of phenolsulphonphthalein was injected into the ventricle at 2:25 P.M. Spinal fluid obtained by lumbar puncture at 2:55 P.M. showed no trace of the dye.

Operation. There was no untoward response to the release of ventricular fluid and on Dec. 15 the glomus of the choroid plexus in the right lateral ventricle was removed as in the previous cases.

Course. The baby made an uneventful recovery. On Dec. 22 (7 days after operation) the ventricular pressure measured 110 mm. of water in the prone position. One cc. of phenolsulphonphthalein was injected at 2:25 P.M. The dye was recovered in the spinal fluid in very definite concentration at 3:00 P.M. It is fair, therefore, to assume that a non-communicating had been converted into a communicating hydrocephalus through a new foramen.*

Dec. 30, 1941. The encephalocele was repaired in the usual way. Occipital brain tissue contained in it was amputated. No part of the ventricle was included. Recovery was uneventful and the baby was discharged in good condition on Jan. 7, 1942.

Feb. 20, 1942. On follow-up examination the baby appeared to be in excellent condition. The anterior fontanelle was soft and depressed and its dimensions had decreased by 1 cm. The greatest circumference of the head had not increased over that at the last admission.

Case 4. P.C., a white male aged 50, was admitted on Sept. 6, 1941. He complained of headache and generalized weakness for 2 years. Three months ago the headache became quite severe and he became bedfast. Three weeks ago he began vomiting and one week ago he had an "unconscious spell."

Other than for disorientation, inability to walk, restricted visual fields and a fine tremor of the tongue there were few signs and no focalizing signs. The eye grounds were normal and the spinal fluid pressure 190 mm. of water. Air was injected into the spinal subarachnoid space. Roentgenograms showed air in the cerebellar, interpeduncular, and chiasmatic cisterns but very little if any in the cortical subarachnoid spaces and none in the ventricles. Ventriculography revealed a marked symmetrical dilatation of the ventricles and dilatation of the 4th ventricle to the foramen of Magendie without other deformity or filling defect. No air gained access to the basilar cisterns or cortical subarachnoid spaces. A cerebellar exploration revealed a normal cerebellum and the cerebellar cistern was filled with clear fluid under tension. The cistern was ruptured and the foramen of Magendie found to be patent. Saline injected into a ventricle flowed freely through the foramen. A section of the arachnoid revealed microscopically the evidence of chronic inflammation.

After this operation he became much clearer mentally and began to walk well. He was discharged on October 18 greatly improved.

For 3 weeks the improvement continued and he then began to lose ground again. He was readmitted on Nov. 25, 1941, and presented about the same findings as on the previous admission. A ventriculogram revealed the same findings as on the first admission and a diagnosis of adhesive arachnitis was made.

Phenolsulphonphthalein test (Nov. 29, 1941). One cc. of the dye was injected into a ventricle. After 20 minutes and also 1 hour no dye could be recovered from the spinal fluid.

Urine was collected at 15-minute intervals but no trace of the dye appeared after 2 hours. The hydrocephalus appeared to have become non-communicating. At the end of 2 hours dye was recovered from the ventricles.

Choroid plexectomy (Dec. 5, 1941). An incision was made through the right occipital lobe into the posterior horn. The glomus of the choroid plexus was dissected away. The laminae of the plexus were definite and when they were severed at their affixed margins, the slit or choroidal fissure created between the fornix and thalamus was obvious. The right ventricle had been emptied of fluid and the moment the plexus had been removed some fluid seeped into the ventricle through the newly made communication. The fluid undoubtedly came from the cisterna venae magnae.

Phenolsulphonphthalein tests (Dec. 11 and Dec. 20, 1941). On each of these two occasions after the choroid plexectomy 1 cc. of the dye was injected into a ventricle. The dye could not be recovered in the spinal fluid in $\frac{1}{2}$ hour but each time it appeared in definite amounts in the urine in $\frac{1}{2}$ hour. On the last occasion (15 days after operation) the ventricular pressure measured 85 mm. of water in the prone position.

* I feel that the opening made through the brain to expose the ventricle in this case and Case 4 is not likely the route of escape of ventricular fluid into the subarachnoid spaces.

Hence, we feel it fair to assume that although the dye did not find its way into the spinal subarachnoid space, ventricular fluid was now being absorbed in the subarachnoid spaces above the tentorium.

The patient's mental status improved slowly but he remained bedridden when discharged 3 weeks after operation.

Follow-up examination. The patient was returned to the hospital on Feb. 17, 1942. His mental status was considerably improved. He sat in a chair most of the day and again was able to walk with a little help.

The ventricular trephines were reopened and the ventricular pressure found to be 60 mm. of water in the prone position. One cc. of phenolsulphonphthalein was injected into a ventricle. Dye was not recovered from the spinal fluid in ½ hour nor in 3½ hours. It appeared in definite concentration in the urine in 2 hours. (The urine output was small during the early part of the test.) It was concluded therefore that if ventricular fluid was being secreted, it was being absorbed.

Test for rate of secretion of ventricular fluid. On Feb. 21, 1942 at 5:00 P.M. the patient was placed on his side and a ventricular needle inserted into the uppermost ventricle. The pressure measured 65 mm. of water. The phenolsulphonphthalein which had been injected 4 days previously had entirely disappeared. The patient remained perfectly quiet while ventricular fluid was collected as it dropped from the needle. Fluid was allowed to drop for 7 minutes until the flow became constant. Then 12 cc. of fluid escaped in a 10-minute period. If this can be considered an average output it amounts to 1,728 cc. in 24 hours.* It is assumed, therefore, that although dye could not be recovered from the spinal fluid after injection into a ventricle, fluid was finding an exit through the choroidal fissure and into the subarachnoid spaces above the tentorium.

DISCUSSION

The fact that a communication can be made from ventricle to subarachnoid space by disrupting the laminae of the choroid plexus (ventriculocisternostomy) provides a relatively simple and effective means of "shunting the circulation." Since the subarachnoid spaces of the tela choroidea and the cisternae into which it empties seem least likely to be involved in the usual obliterating processes, it would appear that these spaces are particularly suitable to receive ventricular fluid. The optimum place of attack on the choroid plexus is at the glomus, for this would establish the shortest route from ventricle to cisterna venae magnae. In cauterizing the choroid plexus one should make a special effort to disrupt its laminae.

In creating any artificial opening the question of possible healing and reclosure is important. Openings made in brain tissue are likely to heal over in time but opening of the choroidal fissure does not involve trauma to brain tissue. Will the rent in the pia arachnoid and ependyma heal? It is only my opinion that such a fistula will tend to remain patent. I mentioned previously that I have disrupted a pia-arachnoid membrane which completely closed the foramen of Magendie in three adult patients. This apparently was the only cause of a marked hydrocephalus. The cure was permanent (6 and 7 years at the time of writing) in the two patients who lived. The third died suddenly of unexplained cause before leaving the hospital.† This would in-

* Some such test as this would indicate to what extent the secretion of ventricular fluid has been curtailed after cauterization of the plexuses in both lateral ventricles.

† Two of these patients, one who lived and is well 7 years to date, and the one who did not survive, are reported in a previous publication.[2]

dicate that the pia-arachnoid opening might be expected to remain indefinitely.

It would probably be much better to first cauterize the glomus with bipolar electrodes and then tear it away.

CONCLUSIONS

Recognizing that the clinical evidence presented here should be augmented I would nevertheless like to make the following points:

(1) Disruption of the laminae of the choroid plexus establishes a communication from a lateral ventricle to the subarachnoid spaces of the brain through the space in the tela choroidea. The term *cisterna venae parvae* is suggested to designate this space. The glomus is probably the optimum place for attack.

(2) This procedure provides a simple and probably most effective means of treating congenital hydrocephalus of whatever cause—both communicating and non-communicating. If hydrocephalus is present, it should be so treated before treating any accompanying malformation. In congenital hydrocephalus the procedure should be carried out by means of a lens system and without removing ventricular fluid. Preoperative ventricular taps should be made in order to tide the patient over any untoward response to the release of increased intracranial pressure.

(3) This procedure is equally applicable to the treatment of acquired hydrocephalus with and without subtentorial neoplasm when such measures are warranted.

(4) It is my opinion that the good results that have been obtained by cauterization of the choroid plexus may have been due partly, if not chiefly, to the unwitting creation of such a fistula through the choroidal fissure rather than to a diminished production of ventricular fluid. It may be well to keep the idea in mind when destroying the plexus.

Addendum

Case 5. M.B., #44-16693, a white girl, age 7 years, was first admitted to the Children's Hospital, Denver, Colorado, on Mar. 2, 1944. She presented the usual signs and symptoms of meningitis. A spinal puncture showed a pressure of 600 mm. of water in the prone position. There were 339 cells per c.mm. (258 polymorphs and 81 lymphocytes). The sugar was 24 mgm. and protein 45 mgm. per 100 cc. Pneumococcus type 3 was cultured from the fluid.

She was treated with heavy doses of sulphadiazine orally and penicillin intraspinally (20,000 units twice daily to a total of 200,000 units). The culture was negative in 24 hours. On Mar. 21, 1944 she was dismissed from the hospital apparently recovered and cured of the meningitis, but with a spinal fluid pressure of 400 mm. of water in the prone position.

Second admission (Mar. 27, 1944). After being home a week she became lethargic and had a generalized convulsion. On return to the hospital she was stuporous and presented a left hemiplegia, papilloedema on both sides and a temperature of 99° F. The spinal fluid pressure was 400 mm. of water in the prone position. The chemistry and cytology of the fluid were essentially normal.

A brain abscess was suspected and pneumoventriculograms were made after injecting air directly into the ventricles. The films showed a marked dilatation of all of the ventricles without shift from their normal location and without filling defect (Fig. 10). The ventricular

system was well visualized to the foramen of Magendie. There was evidence that air escaped through the latter, for it appeared in the cerebellar cistern, cervical canal and pontine cistern. There was no evidence whatever of air in the cortical subarachnoid spaces or distal to the pontine cistern. Upon this evidence a diagnosis of acute communicating hydrocephalus was made. The cause of the obstruction was an adhesive arachnitis at the base of the brain following the pneumococcic meningitis.

Spinal punctures were repeatedly done for 10 days and the spinal fluid allowed to escape each time until the pressure dropped to 100 mm. of water or less. The pressure always returned

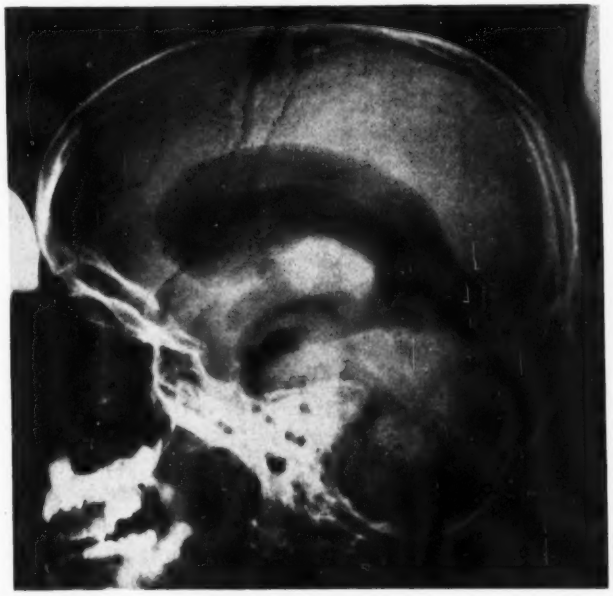

FIG. 10. Case 5. Post-meningitic hydrocephalus. Lateral ventriculogram. The entire ventricular system is dilated but all parts are in normal position. The 3rd ventricle, aqueduct of Sylvius and 4th ventricle are well visualized. Air is visualized in the cisterna pontis indicating that the foramen of Magendie is patent. Air did not progress any further up the cortical subarachnoid spaces than the cisterna pontis, however, indicating an adhesive arachnitis as a result of the meningitis.

to 400 however in a 2-day period. It appeared that the obstruction was complete and that the obstructive hydrocephalus had no tendency to resolve spontaneously.

Operation (April 6, 1944). With the hope that the subarachnoid spaces above the level of the cerebellar tentorium were not obstructed, it was decided that a ventriculo-cisternostomy should be attempted. An incision was made into the posterior horn of the right ventricle and under direct vision the glomus of the choroid plexus was dissected away as close to the fornix as possible.

Course. The patient made slow but continued improvement in both mental and physical status and the spinal pressure resumed a normal figure. She was dismissed from the hospital in 3 weeks.

Third admission (Oct. 13, 1944). Following the last discharge from the hospital the child gained weight and recovered fully so that she was admitted to school. There were occasional petit mal seizures, however, and one day prior to this admission she had a generalized seizure.

The general and neurological examination gave essentially normal findings. The papilloedema had subsided and the spinal fluid pressure was 125 mm. of water in the prone position. She was discharged in 2 days.

Subsequent course. The patient has been reexamined from time to time and has maintained a healthy state mentally and physically except for an occasional seizure (Fig. 11). The last examination was made on Jan. 15, 1946, 21 months after the operation, and spinal fluid pressure was found to be 124 mm.

Discussion. In respect to this case, I feel the following inferences to be sound: (1) Had operative interference not been made the post-infectious acute hydrocephalus would not have resolved and the patient would likely have succumbed. (2) The restoration of spinal fluid pressure to normal was not due to the incision made into the brain. If this were true, hydrocephalus would not present the almost insuperable problem that it does. (3) It is not likely that the removal of choroid plexus in one lateral ventricle would restore the pressure from 400 mm. to normal merely because the output of ventricular fluid is lessened. And this especially since the clinical course, the spinal fluid pressure readings and the ventriculogram before operation indicated that the obstruction to the intracranial circulation was complete. (4) Hence it is fair to assume that the type of fistula described in this paper was established between the right lateral ventricle and the cisterns of the great and small veins of Galen. The fluid then having access to all available subarachnoid spaces distal to the basilar arachnoid obstruction, found sufficiently rapid absorption to maintain normal circulation. (5) Twenty-one months is a fair test of time to indicate that such a fistula does not have a tendency to heal but has a natural tendency to remain patent. (6) The acquired tendency to epileptic seizures could be due to the post-infectious adhesive arachnitis or to the effects of the high pressure imposed upon the brain throughout the duration of the acute hydrocephalus. Since seizures preceded the operation, they were probably not the result of it.

FIG. 11. Case 5. Patient two years after a ventriculocisternostomy as described in the case report.

With the advent of the sulpha drugs and penicillin, cures from pyogenic meningitis are not uncommon. As in the case presented here, however, an obstructive arachnitis with a partial or complete obstructive hydrocephalus may become a problematical sequela in some cases. The pneumococcus in particular, because it is associated with a tenacious exudate, is likely to cause a permanent obstruction to the circulation of cerebrospinal fluid through the basilar cisternae and hence block its path to the indispensable absorbing system in the cortical subarachnoid spaces.

If obstruction of the basilar subarachnoid spaces does obtain following meningitis, it does not necessarily follow that the upper subarachnoid spaces (those above the level of the tentorium and corpus callosum) will be involved to the same extent. Postmortem examination in such cases indicates that the exudate is concentrated at the base of the brain with very little in the upper subarachnoid spaces.

If post-infectious obstruction to the circulation and absorption of cerebrospinal fluid is complete, it is a serious matter. It is my opinion that an acute and complete obstructive hydrocephalus is incompatible with life and if such a state is allowed to exist for an appreciable length of time before it is alleviated, the brain will suffer damage irreparably and progressively.

The method of treatment described here appears to be promising and is the only procedure to my knowledge that offers any hope of success at the present time. The factors involved in the treatment of acquired obstructive hydrocephalus differ considerably from those of congenital hydrocephalus and are much more favorable.

Post-meningitic hydrocephalus may now become of greater significance and merit more than passing consideration. Hence, the renewed interest in the application of these principles in the treatment of hydrocephalus.

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CASE REPORT AND TECHNICAL NOTES

MUCOCELE OF THE SPHENOID SINUS

REPORT OF A CASE WITH AUTOPSY FINDINGS

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Since Garreau[8] first called attention to the condition in 1881 and Rollet[17] introduced the term mucocele in 1896, this affection of the accessory nasal sinuses has been followed with considerable interest. Within more recent years, its relatively frequent occurrence has been more appreciated. As early as 1909 Gerber[11] collected 178 cases and Howarth[11,12] found nearly 300 by 1921 and thought there were many more.

Mucoceles of the sphenoid sinus on the other hand have been described quite infrequently. Among the few appearing in the literature are those of Rolland, Knapp, Polyak,[16] Hajek,[9] Benjamins,[1] Litwinowicz,[13] Rhese,[16] Van der Hoeve,[20] Meisels,[14] Schüller,[18] O'Shea,[15] Cavina,[4] de Francesco,[6] Fornari,[7] Berendes,[2] and Bilchick.[3] Including others which may have been overlooked, it seems reasonable to assume that not many more than 25 have been published in the medical literature at this time. Some of the cases reported have been little more than brief notes, and probably many of them cannot be accepted as sphenoid mucoceles without criticism.

Mucocele is defined frequently as an accumulation and retention within a sinus of mucoid material or secretion resulting from continued or periodic closure of its ostium with gradual thinning, distension, or actual erosion of one or more of its walls. When the contents of the cavity are purulent in character, the condition is referred to as a suppurating mucocele or pyocele.

In the more commonly occurring frontal and ethmoidal mucoceles, the clinical features are orbital in character with proptosis and other signs leading to the diagnosis. The sphenoid sinus mucocele, however, presents a difficult problem diagnostically. This can be appreciated from reviewing those described in the literature, as well as from the experience gained in the observation of the unusual and interesting case we are reporting at this time. We have, moreover, been unable to discover any other autopsy examinations in this condition.

REPORT OF CASE

Mr. L.H. (Case #20034), aged 54, former accountant by occupation, was referred to the Good Hope Clinic July 21, 1938 by Dr. C. Morley Sellery. He gave the history of having suffered with sinusitis and associated left-sided headaches since 1920. He was first operated upon in 1923, when the left frontal sinus was opened and the left middle turbinate removed. In 1931 an operation was performed on the left antrum, and a Caldwell-Luc procedure done in 1932. In addition the patient had numerous sinus treatments. Although colds were infrequent, he noticed an almost constant, thin, whitish nasal discharge. With his not infrequent attacks of sinusitis, this seemed to become thick and rather yellowish. He also discovered that his headaches, which were invariably left frontal with radiation to the left side of the head, were worse with the sinus attacks, and were relieved by the flow of the nasal discharge. During July 1937 he experienced his first spell of severe left frontal and occipital headache, which he presumed was the result of an attack of influenza. During this time he felt weak and incapacitated. A second episode occurred about 6 weeks later, but this time his temperature was subnormal rather than elevated. A third episode, which took place during December 1937, lasted 2 weeks. A very similar attack of headache and exhaustion occurred about February 1938. In May 1938 he again had a spell of headache, this time preceded by a chill and an incident in which he lost consciousness. He was in bed for about 4 weeks.

The family history was irrelevant. The past history revealed that the patient's general health had been good prior to his symptoms which began about 1920. He had the usual childhood diseases including a severe case of diphtheria at the age of 6 years, following which he had a bilateral otitis media. Since that time he had been deaf in the left ear and hearing had been impaired in the right. The past history was otherwise without significance.

Examination. The patient was an apathetic man, somewhat undernourished, weighing 124 pounds, 5 feet 6 inches tall, with a pale and sallow complexion, mentally clear and cooperative. There was no stiffness of the neck. A large perforation was seen in the right tympanic membrane which appeared to be in contact with the promontory. The left ear drum was also adherent to the promontory. Nothing unusual was discovered in the examination of the heart, lungs, abdomen, or extremities. In the neurological examination he was unable to identify camphor or coffee in the left nostril, but he did so normally in the right. The watch tick was not heard when the watch was pressed against the left ear, but was heard at a distance of 4 inches from the right ear. Air conduction was greater than bone conduction in the right ear; while bone conduction was heard in the left ear, but no air conduction was present. The Weber was not referred. The general neurological examination disclosed no abnormal findings.

Examination in the otolaryngological clinic showed both antra to be dark on illumination and the frontals to be translucent. Nasal examination revealed a pale mucous membrane with no polyps present. The left ostium of the sphenoid appeared normal. The left antrum was irrigated and the fluid returned clear. Caloric vestibular tests at this time showed no response from the left side, and a subnormal response from the right vertical canals. The impression was that the left ear was not responsive to stimulation.

Ophthalmological consultation disclosed a visual acuity of 20/20 in each eye. Funduscopic examination revealed a picture of healed bilateral choroiditis of the perimacular area.

Urinalysis was not unusual. The hemoglobin was 82 per cent, the erythrocyte count 4,510,000, with a color index of 0.91, and leukocyte count 8,400. The differential examination showed nothing peculiar. The blood Wassermann was negative, non-protein nitrogen 38 mg. per cent, urea nitrogen 19 mg. and serum sodium 307 mg. The basal metabolic rate was recorded as minus 26.

Roentgenograms of the skull were interesting in disclosing the presence of two irregularly shaped translucent areas in the occipital bone which seemed suggestive of a diploic epidermoid or cholesteatoma. The pineal gland was calcified but showed no displacement. The posterior clinoids were completely wiped out, and the left anterior clinoid process appeared to be eroded. The sella was also enlarged suggesting a tumor in this location. It was believed he might have a cholesteatoma in the occipital region and possibly in the proximity of the third ventricle as well. Hospitalization for ventriculography and probably craniotomy was recommended.

Operation I. The patient was admitted to the Hospital of the Good Samaritan and operated upon Feb. 16, 1939. A ventriculogram revealed ventricles of normal size with no significant displacement. Because of the peculiar markings in the occipital bone, it was believed that these should nevertheless be explored and the etiology determined. A suboccipital craniotomy was carried out exposing these mentioned areas, which seemed obviously due to arachnoidal abnormalities. The dura was opened and no evidence of a cholesteatoma discovered. A piece of tissue was removed from the rarefied areas including the dura in the region, and microscopic examination was reported as showing principally fibrous tissue with numerous thin-walled vessels.

Course. He recovered uneventfully, but on the third postoperative day vision of his left eye was markedly impaired. This failed to improve and pointed to pathology in the pituitary area. Roentgenograms of the skull were again reviewed and the erosion of the sella noted as well as what appeared to be the calcification of a suprasellar cyst. Further questioning brought out the fact that on several previous occasions the patient had noted diplopia or blurred vision.

Operation II. The patient was reoperated upon Mar. 11, 1939 with the preoperative diagnosis of suprasellar meningioma or cyst. A left frontotemporal osteoplastic craniotomy was carried out with this in view. The frontal lobe was retracted exposing the pituitary fossa. An

unusual picture was uncovered at the base of the skull in this region (Fig. 1). An irregular, bluish elevation some 4 cm. in diameter was noted. There appeared to be no pulsation. Palpation gave the impression of thinned and parchment-like bone, which was thought to be possibly some kind of aneurysmal dilatation. It seemed obviously the cause of the patient's symptoms. The area was aspirated carefully with the removal of about 9 cc. of thick, greenish-brown material. This was taken to the laboratory where a smear examination showed no organisms. The cystic area was partially opened and its contents suctioned out. The cystic area seemed to extend into the sphenoid sinus. It was presumed the condition represented an infection of the sphenoid sinus and meningitis was feared as a subsequent sequela. The cyst wall was approximated and a piece of muscle covered over the small defect. Closure was then carried out in the usual manner.

The laboratory described the material obtained at operation as a thick, olive-colored fluid. Microscopic examination of a wet-mount preparation showed a few cholesterin crystals, numerous pus and a few red blood cells. Some of the cells appeared to be filled with highly refractive granules. A gram stain of a direct smear from the fluid disclosed a few polynuclear leukocytes, an occasional mononuclear cell, but no organisms. Brain broth inoculated with the fluid showed no growth after an incubation of 48 hours.

Course. Following a critical period during the first 48 hours, the patient made a good recovery and was noticeably relieved of his headaches. The vision of the left eye, however, remained unchanged. Examination Mar. 30, 1939 by Dr. C. L. Weeks disclosed the previously described perimacular chorioretinitis. There was an increased pallor generally with accentuation of the central area, and the fields taken revealed, as anticipated by the funduscopic picture, light perception only.

Operation III. In view of the operative findings, an intranasal sphenoid investigation was carried out April 18, 1939. The face of the sphenoid was exposed by fracturing back the posterior end of the middle turbinate. The entire sphenoid area was found to be a membranous wall, or at least, if any bone was present, it was so attenuated that it offered no resistance. A probe passed into the cavity a little over 3 cm. in an anteroposterior direction. Several cc. of thick, brick-red material were removed by means of a large-calibre needle. A gram stain of a direct smear of this showed an occasional mononuclear leukocyte, but no organisms. Broth cultures inoculated with a loop of the fluid revealed a growth of gram positive cocci in pairs and groups. Subcultures from the broth grew *Staphylococcus albus*. Following this, the entire anterior sphenoid region assumed a distinct pulsation. No further operative intervention was believed advisable at this time.

Course. The patient was discharged from the hospital April 21, 1939 and was followed in the Good Hope Clinic. The condition of the left eye remained unchanged.

When he was examined May 4, 1939, the fullness in the sphenoid region was found to be distinctly less. Light perception only was evident in the left eye. No essential changes were observed on his visit May 25, 1939. On Jan. 3, 1940 no pulsation was seen in the right sphenoid area and some fullness was apparent. There was polypoid degeneration in that region. On the left side the posterior border of the septum was crowded against the posterior end of the inferior turbinate and the sphenoid fullness was much diminished. On Feb. 1, 1940 the patient complained of a full feeling in his head and mucous dripping from the throat. No pulsation was seen in the sphenoid region. On Feb. 27, 1940, about 3 cc. of brick-red, thick exudate were removed from the sphenoid cyst with a large-calibre needle, and on Mar. 5, 1940 there appeared to be some seepage from this opening. The cyst was again aspirated and about 3 cc. of the same material evacuated. On Mar. 12, 1940 there appeared to be a decided decrease in the bulging of the sphenoid area. There was a small amount of drainage from the site of the preceding puncture. About 2 drams of heavy brownish material were removed from the cyst on Mar. 20, 1940, and a very small amount of material was withdrawn June 4, 1940. There was no change in his vision or in the visual fields.

During the preceding week the patient had been troubled with his old headaches. A small amount of light-colored material was aspirated July 9, 1940. The patient continued having his headaches, which became more severe; in addition, he began experiencing frequent

episodes of drowsiness. He also noticed a swelling of his left eye which subsided after a period of about 7 days. According to his statement, he had had similar attacks in the past. He was admitted again to the Hospital of the Good Samaritan July 13, 1940. On the morning of the 16th he had a Jacksonian convulsive seizure involving the right side of the body. He was approaching a stage of status epilepticus when the seizures were brought under control by means of 7½ gr. of sodium amytal administered intravenously. A lumbar puncture done shortly afterward disclosed a pressure of 70 mm. of spinal fluid. The cell count was 105 with about 86 per cent polymorphonuclears and 14 per cent mononuclears. The total protein was 135

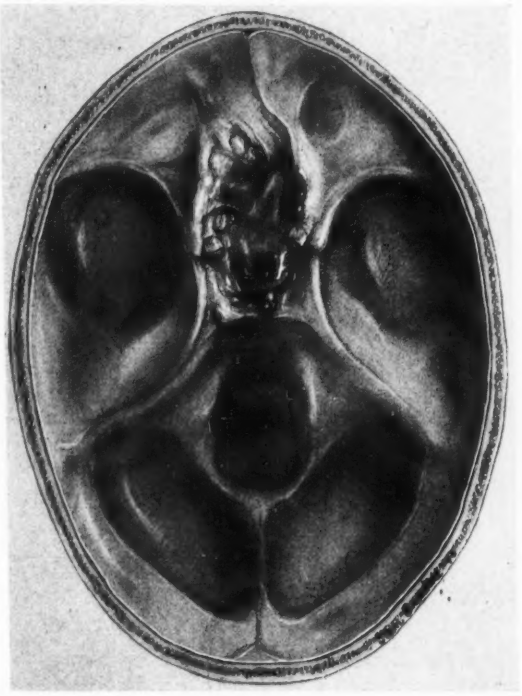

FIG. 1. Medical illustrator's picture of the appearance of the roof of the sphenoid mucocele shown at postmortem.

mg. per cent. No bacteria were found in the stained smear and none was grown on culture. The temperature the previous day and prior to the convulsion was elevated to 104°. On the 18th the patient seemed very much improved, the temperature had approached a normal level, he no longer complained of headache, and he seemed anxious for food. The following day, however, he again complained of headache and elevation of the temperature was evident. Stiffness of the neck continued with bilaterally positive Kernig's sign. It was believed that he had a low-grade meningitis as a result of the rupture of the sphenoid sinus mucocele. His course continued somewhat the same with a spiking temperature. At times he appeared rational, while on other occasions he was delirious and semicomatose.

On Aug. 2, 1940, although he appeared fairly clear mentally, his neck continued to be quite stiff. A lumbar puncture was done which revealed a pressure of 70 mm. of spinal fluid. About 10 cc. of slightly turbid fluid were removed. This showed no growth on culture after an incu-

bation of 72 hours. His course varied with occasions during which he was quite drowsy and weak, and other instances when he was able to sit up and take nourishment, but in general, his memory was poor and his activities were like those seen in frontal lobe conditions. He had little realization of the seriousness of his illness.

Radiographs taken of the skull at this time showed that the left sphenoid was very hazy and that the left frontal sinus was not as clear as the right. Both antra appeared to be slightly cloudy with evidence of mucous membrane thickening, especially on the left. The posterior ethmoid cells could not be made out at all on the left, and only two small infra-orbital ones were seen to be outlined clearly. Marked enlargement and distortion of the left orbital fissure

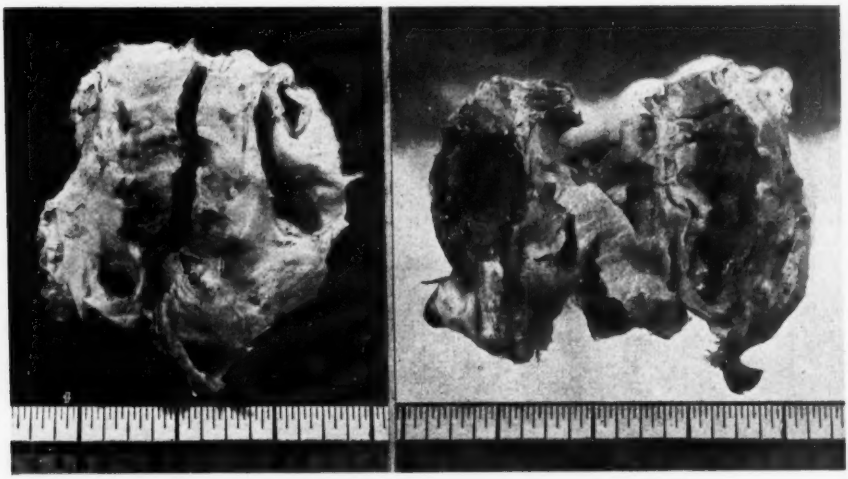

FIG. 2 (*left*). Photograph of the sphenoid region removed *en masse* at postmortem, showing its roof. An incision has been made through its center to show the size of the cavity as illustrated in Fig. 3.

FIG. 3 (*right*). Photograph of the sphenoid mucocele sectioned sagitally, showing the interior of each half of the cyst.

were evident. By Sept. 5, 1940 he was rather unresponsive and there was a considerable quantity of mucopurulent discharge from his nasopharynx. Some spasticity of the right arm and leg was noted at this time. By Sept. 8, 1940 the patient slipped into a condition of coma and died on Sept. 9, 1940. Permission for autopsy examination was obtained.

AUTOPSY EXAMINATION

The autopsy was performed by Dr. Roy W. Hammack, who reported the following findings: The body was that of an adult male in a state of poor nutrition. A well-healed surgical incision in the left frontoparietal region was evident. Removal of the scalp revealed the bone flap fixed rigidly in place, and at several points between the trephine holes there appeared to be bony union. The convolutions of the brain were flattened. The left frontal lobe was much larger than the right, and its inferior surface near the midline was adherent to the floor of the skull. Coronal sectioning revealed a large abscess of the left frontal lobe beneath the ventricle, the diameters of which were 4.5 cm. horizontally, 3 cm. vertically, and 5 cm. anteroposteriorly. It was demarcated from the surrounding tissue by a fairly definite wall and pyogenic membrane. A gram stain of a smear from the abscess showed numerous pus cells, and few gram positive cocci, occurring singly, in pairs, and occasionally in short chains.

The entire sphenoid region was removed *en masse* for study. As shown photographically (Figs. 2, 3), the mucocele of the sphenoid sinus measured 5 cm. in its greatest anteroposterior, 2.5 cm. in its greatest vertical, and 2 cm. in its greatest lateral diameters. The medical illustrator's drawing of the sphenoid mucocele is shown in Figs. 1 and 4. Very little bone remained on its roof, especially so on the left side where replacement by fibrous tissue had occurred. In spite of the anterior extension of the sphenoid mucocele, the ethmoid region appeared intact. The left optic nerve was noticeably compressed and flattened. The posterior clinoid processes were quite flattened, the anterior less so, but that on the left side was flattened considerably in comparison with the right. The groove of the sella turcica was markedly flattened and broadened, and the hypophysis had been pushed to the right of the midline. It was also flattened and adherent to the base.

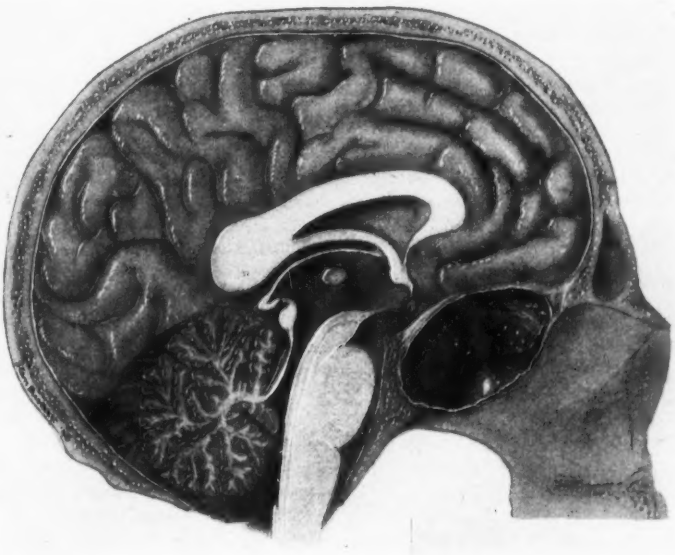

FIG. 4. Medical illustrator's drawing of a sagittal section of the head, showing the mucocele cavity.

Microscopic examination of a section taken from the wall of the cyst revealed an inner layer of fibrous connective tissue containing several areas of small-cell infiltration of polynuclear leukocytes and spindle cells. Below this, along the lining of the sinus, the blood vessels were more numerous as was the infiltration of polymorphonuclear leukocytes and additionally numerous macrophages, many of which contained cholesterin crystals. Rarely a few groups of flattened mucous glands were also observed.

Both lungs were large, heavy, and moist. The posterior portions were congested markedly. In the posterior part of the left lower lobe were several nodules of bronchopneumonia which were gray on section. In the right lower lobe there were fewer and smaller nodules. The heart was normal in appearance, and no gross lesions were discovered in the examination of the abdominal viscera.

DISCUSSION

As can be appreciated from cases reported in the literature[3,5,15,16,20] the diagnosis of sphenoid sinus mucocele is difficult, has seldom been made preoperatively, and often has been discovered inadvertently. Berendes[2] mentioned there is usually a visible nasal secretion and a forward bulging of the sphenoid wall, which, if appreciated, may lead to the diagnosis. His case was discovered unexpectedly as a result of a nasal septum resection. O'Shea's case revealed an exophthalmos.[15] Visual disturbances are not uncommon and may be transient. Van der Hoeve[20] wrote on the subject of atrophy of the optic nerves, and Litwinowicz[13] upon the occurrence of amaurosis some weeks after the beginning of the headache. Headache is a usual symptom. Meisels[14] pointed out the value of roentgenographic findings in the diagnosis, which has either been overlooked or not appreciated in many of the other cases reported with the exception of those of Hajek,[9] Benjamins,[1] Gerber,[11,12] Van der Hoeve,[20] and Schüller.[18] Schüller emphasized that on the basis of the roentgenographic pictures, the differentiation between sphenoid sinus mucocele and intra- or subsellar hypophyseal cysts was very difficult. This would seem apparent from the findings in the case we have presented. There is a wide variance in the age at which the mucocele may develop. As a result of his investigations, Turner[19] stressed the frequency of this condition in youth and early adult life. In Howarth's[11,12] series, however, most of the patients were over 60. It is evident from the literature that the age of the patient is too variable a factor to be of any value in the diagnosis. The condition, moreover, seems to occur in both sexes with equal frequency. It is, of course, difficult to come to a satisfactory conclusion concerning the age of development, for the reason that the progress is markedly slow and innocent, essentially chronic, and some patients have gone as long as 18 years after the appearance of a swelling before considering it necessary to consult a doctor. Even in the less easily diagnosed and quite infrequently observed sphenoid mucoceles, it is difficult to know the period of the onset of the condition.

ETIOLOGY

The origin of mucoceles of the accessory sinuses has been the subject of much controversy; and although in certain cases the explanation has seemed reasonable, no proposed theory for the condition generally has been without criticism. Injury has been suggested by Killian and others[12] as a possible explanation, and isolated cases have been presented on this basis, but in Logan Turner's[19] series of ten cases there was no instance of it and he was inclined to discount the theory. In view of the frequent occurrence of head injuries, it seems strange the condition is not quite common.

Occlusion of the ostium from chronic inflammatory changes or from the pressure of a tumor has received considerable attention as a possible explanation of the cause. Congenital narrowing of the duct rendering a catarrhal inflammatory obstruction is also given as a likely cause as well as septal deviation, and too, congenital absence of a communication from the nose and the frontal sinus.

Other writers[1,5] have inclined to the theory of a cystic dilatation of one or more mucous glands, or a cystic degeneration of the mucosa of the polyps therein. Schüller[18] pointed out the possibility of the development of an hypophyseal cyst from the inner or under half of the sella turcica itself. This conception is in agreement with the ideas of Erdheim, Haberfeld, Lœgaard, Kraus, and others, that not infrequently the inner half of the sphenoid-body-rests of the hypophyseal tissue can furnish the point of origin of the cystic tumor. No adequate proof that the condition is a retention cyst of the sphenoid sinus or the hypophysis seems available from the findings at operation or from the material obtained at operation. In view of the similarity to other mucoceles of the accessory sinuses, however, the hypophyseal basis does not seem very convincing.

In regard to the occlusion of the ostium as the cause of the condition, it has been found that not every closure of an accessory sinus leads to a mucocele.[16] Moreover, in Turner's series,[19] the nasofrontal duct was found to be closed in four cases and open in three. Heatly[10] pointed out from a review of the literature, that the duct was not completely closed in all cases. Dabney[5] was of the opinion that although at times closure of the duct exerted a

causative influence, it was not an essential condition, inasmuch as some of the patients revealed a perfectly patulous duct. As he mentioned, one of his patients had a canal wide enough to admit a large ethmoid curette, and the duct, moreover, had been open for years as a result of repeated intranasal operations on the anterior ethmoid.

Rhese[16] believed a mucocele did not need to be an expression of chronic inflammatory process. It would seem, moreover, that for such to be the case, mucoceles would be much more frequent in occurrence. As Howarth[11,12] has expressed it, "It may be wondered why everyone who gets a nasal catarrh or sustains a severe black eye does not develop a mucocele."

One must be forced to conclude that the onset of these cases is often so gradual and the course so slow that it is difficult to assign a definite cause in an individual case, much less to accept any etiological factor which is common to all of them.

PATHOLOGY

In a few of the published cases of mucocele, the microscopic changes seen in portions of the removed wall have been mentioned. Turner[19] described the detailed account given by Onodi. He found that although the ciliated epithelium was preserved upon some parts of the surface, in others it was apparently replaced by layers of pavement cells. Beneath the epithelium there was a fibrous connective-tissue layer with small-cell infiltration, consisting of spindle cells and polymorphonuclear leukocytes. A number of blood vessels were present, and scattered groups of mucous glands were also observed. External to the connective-tissue layer, there was a thin lamella of bone covered with periosteum consisting of three to four rows of cells and loosely connected with the mucous membrane. The periosteum on both sides of the layer of bone was rich in cells, those next to the bone being of the nature of osteoblasts. The wall of the medullary spaces, as well as the surface of the osseous layer, was smooth, and only in places was there evidence of absorption of the lacunae.

According to Heatly[10] the studies of Zuckerkandl and Onodi have shown mucous glands only at intervals. He pointed out, moreover, that in the patients with mucocele examined by Onodi and others, changes of low-grade inflammatory nature were present both in the lining mucous membrane and in the underlying bone. The lining mucous membrane was usually thin in comparison with the thickened and often polypoid mucous membrane observed in cases of chronic suppuration.

Turner[19] explained the increased accumulation of the secretion in the mucocele as a result of the increased function of the glands consequent to inflammatory changes. At a later stage he felt there were atrophic changes in the lining membrane resulting from the pressure of the accumulation within the cavity and in these changes the glands participate.

In Benjamins'[1] case of sphenoid mucocele there were numerous complex glands which were cystically dilated. The bony tissue ended abruptly and went over uninterruptedly into the connective tissue of the submucosa, which was markedly thickened and contained many thick or dilated blood-filled capillaries.

Rhese's[16] case of sphenoid mucocele failed to reveal any evidence of inflammatory process.

The character of the material obtained from mucoceles of the accessory sinuses has been variously described. Van der Hoeve[20] described the material in his case as brownish-green tenacious mucoid substance without odor and containing many cholesterol crystals. In O'Shea's case[15] it was found to be a dark, reddish-brown, glairy fluid. It has been also mentioned as being thin, syrupy, and resembling Hollandaise sauce, while Turner[19] stated that in the majority of cases the contents are of a thick, tenacious, mucoid consistency. He found in some cases that the fluid was clear and serous, presenting none of the physical characteristics of mucus but more resembling cerebrospinal fluid. He also mentioned that in several cases it has been described as having a greenish appearance and hence the readiness of mistaking it for pus.

TREATMENT

There have been so few sphenoid sinus mucoceles that the method of treatment cannot be discussed adequately. According to Dabney,[6] in the treatment of the other accessory sinus

mucoceles therapy should be directed toward establishing a large permanent communication with the nose. Attempts to obliterate the cavity or to preserve it without establishing adequate intranasal drainage have repeatedly proven unsuccessful. Berendes[2] mentioned in regard to the sphenoid sinus mucocele that treatment must aim toward the creation of an outflow or passage. With the obstructed condition of the nose, he felt that the most suitable method would seem to be an operation through the ethmoid, and according to his reviews of the subject, the best results were obtained in this way. It would seem that adequate drainage would offer the best possible result, but even here one wonders about the subsequent infection of the cystic cavity and about the ultimate result over a period of time.

As is so often the case, in retrospect had the possibilities of a sphenoid mucocele been under consideration, such a diagnosis might have occurred or have been presumed in our case, and the diagnosis verified by a nasal approach. In view of the essentially normal findings on the nasal examination in the clinic and the unusual markings in the occipital bone, which were thought to be due to cholesteatomas, it is not surprising that the diagnosis was thrown farther away from any consideration of accessory nasal sinus disease. With the sudden loss of vision of the left eye following occipital exploration, a suprasellar cyst seemed most likely, and the x-ray films were certainly suggestive of this. Such a condition is much more frequently encountered than sphenoid sinus mucocele, and intracranial exploration seemed a logical procedure. It was at this time that the true nature of the disease was discovered. With the opening of the parchment-like wall of the cyst, meningitis was feared as a complication, but failed to occur. Interestingly the material showed no organisms and no growth was obtained on culture. In view of the opening of the cyst, subsequent treatment of the large sphenoid mucocele by drainage via the endonasal route was complicated, for infection from the sinuses would probably lead to meningitis or abscess formation, as was found to be true in our case. When the material was removed at this time *Staphylococcus albus* was grown on culture.

Interestingly, the pulsation of the sphenoid region noted after aspiration of the mucocele has been described in other reports. O'Shea[15] had the impression he had opened an aneurysm. He described the pulsation as being very marked with fluid filling the entire operative area. He accounted for the pulsation from the fact that the extensive exposure of the dura, together with the fact that the large vessels and cavernous sinus were in close proximity to the mucocele. Rhese[16] also described the pulsation of the inner portion of the sphenoid sinus when the same had been exposed.

In retrospect our patient had most of the symptoms of sphenoid sinus mucocele. Visual disturbance and optic atrophy have often been found. The history was ultimately obtained that the patient had had previous episodes of swelling of the left eye. The amaurosis occurred after his first operation, but the ophthalmological examination did reveal a picture previously of a healed bilateral choroiditis of the perimacular area, and the patient had noted diplopia and blurred vision on several occasions. He had headaches, which is a usual symptom, and in his case it was noted that these subsided following flow of a nasal discharge and were worse with his sinus attacks. His episodes of unconsciousness were undoubtedly the result of the increased pressure in the region of the third ventricle. Schüller[18] also reported an episode of unconsciousness in his case. As in Schüller's case, the x-ray films in our case would be differentiated with difficulty from those of sphenoid sinus mucocele and intra- or subsellar hypophyseal cysts.

SUMMARY

A case of a large mucocele of the sphenoid sinus with subsequent autopsy examination has been reported.

While mucoceles of the other accessory nasal sinuses have been described not infrequently, those of the sphenoid sinus have occurred rarely and it is believed not more than 25 have made their appearance in medical literature at this time.

In the more commonly occurring frontal and ethmoidal mucoceles, the clinical features are orbital in character, with proptosis and other signs leading to the diagnosis, but in contrast the diagnosis of sphenoid sinus mucocele is difficult, has seldom been made preopera-

tively, and often has been discovered inadvertently. Its differentiation, moreover, from hypophyseal cysts may be quite difficult.

The origin of mucoceles of the accessory sinuses has been the subject of much controversy and no presented theory has been received without logical criticism.

A brief discussion of the pathology and treatment of the condition is given.

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A TECHNIQUE FOR CHARTING NEUROSURGICAL LESIONS

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At the present time there is no standard technique for charting neurosurgical lesions comparable, for example, to that for charting visual fields. An artist's sketch or drawing of the operative field, the surgeon's sketch, usually reconstructed from memory, and, in some instances, photography have variously been used to communicate the grosser details of

neurosurgical procedures. Still-photography usually provides no notion of the relative extent of the operative field, due to the presence of surgical wrappings, although it may yield an excellent degree of detail even as to color.

While working in close cooperation with neurosurgeons during the past ten years,* the writer has become impressed with the need for a simple, convenient, and accurate technique of charting neurosurgical lesions, preferably at the time of operation, for purposes of permanent record.[2,3] It is believed that the technique described below supplies this need. Positive features of the technique include (a) a standard way of recording the neuro-anatomical results of brain operations as part of the medical history of the patient, (b) a convenient type of record for purposes of publication, and (c) an accurate method of charting for purposes of quantitative comparison with other types of data. To obviate possible objections that the technique is unduly consuming of the surgeon's time, brain diagrams have been prepared as standard charts on which the neurosurgical lesion is plotted directly in three dimensions during the terminal stage of the operation. This procedure requires about five minutes of operating-room time to yield the basis for a relatively accurate and permanent map which may be filed as part of the surgical history. No alteration in operating-room procedure is necessary since the charts are printed on all-rag paper stock which under test was found to stand up under one autoclaving without distortion. Many neurosurgeons routinely measure extirpated areas with a sterile brain-puncture needle or ruler. Such measurements can now be translated directly to a sterilized chart by the surgeon's assistant.

BRAIN-LESION CHARTS

A sample brain-lesion chart for left orientation, on which has been diagrammed a left prefrontal lobectomy, is shown in Fig. 1. The originals are printed on $8\frac{1}{2} \times 11$ inch forms. The gray areas, indicating extirpated areas, were prepared by applying No. 64 Zip-a-tone† engraving screen material directly to each brain diagram.

The charts are available at cost from The University of Chicago Press, Chicago 37, Illinois, for either a right-sided or left-sided orientation. Each chart provides four basic diagrammatic views of the brain reduced to one-third of life size (a superior, a lateral, a ventral, and a median-sagittal view). These views, to which internal and external orienting landmarks have been added, are based upon the normal hemisphere of the brain studied in great histological detail by v. Economo and Koskinas.[1] They were drawn approximately to life size in the studio of the well-known medical illustrator, Mr. Tom Jones, who worked in close consultation with Dr. Percival Bailey.

SCHEMATIC BRAIN MODEL

Where careful quantitative and qualitative studies of the behavior of neurosurgical patients are undertaken, it becomes desirable to specify accurately the location and the extent of each brain lesion in quantitative terms *antemortem*.[2] To aid in this task, a schematic brain model has been constructed. A photograph of the model is shown in Fig. 2. The model is used in connection with the brain-lesion charts described above. In constructing the model, the original drawings supplied by Mr. Jones for the brain-lesion charts were copied photographically without reduction onto translite film, to which grid lines were added. The four views were then mounted under sheets of $\frac{1}{8}$ inch thick plexiglass which were in turn mounted around the four edges of $7 \times 7 \times \frac{1}{4}$ inch sheets of black bakelite. The rectangular box, approximately $10 \times 7 \times 7$ inches, thus formed, was then suspended on an 18-inch, 15-watt white fluorescent lamp, passed through the horizontal axis and about which the model may rotate freely. Any of the four diagrams may thus be brought into view by manual rotation of the model.

* The writer is indebted for helpful suggestions from Dr. A. Earl Walker, Dr. Jack Woolf, and Dr. Percival Bailey.

† This material may be purchased at nominal cost from The Paratone Company, 343 South Dearborn Street, Chicago, Illinois.

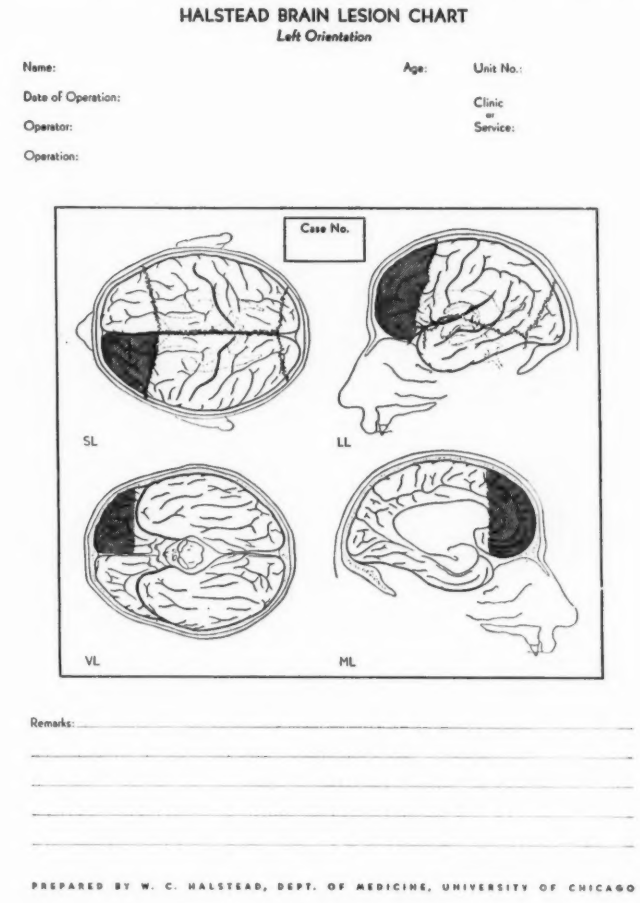

FIG. 1. Sample brain-lesion chart for left orientation. A hypothetical left prefrontal lobectomy is represented diagrammatically in three dimensions by the shaded areas.

The fluorescent lamp supplies cold, diffuse, internal illumination of the model. The brain model is supported by an open frame-work which rises 33 inches above the floor to convenient viewing height. For easy reference, a rear-illuminated lateral view of the architectonic map of v. Economo and Koskinas,[1] also copied on translite film, is mounted below the schematic brain model.

In use, a lateral skull x-ray view of the patient is placed in contact with the lateral view of the internally illuminated brain model. Significant deviations in contour of the inner table of the calvarium from the former are then measured directly by means of a millimeter scale and recorded as plus or minus quantities on graph paper. A smoothed curve is then drawn through these points. Its open extent thus bounds the fronto-occipital dimension of the hemisphere. A maximal vertical dimension is similarly obtained from the skull plate. The product

of these two dimensions from the skull plate is used as the numerator in a fraction, the denominator of which is the similar value taken as a constant from the brain model.

Deviation of this fraction from unity is used as a percentage size correction factor in translating the lesion measured at the time of operation to a final brain-lesion chart. The extent of the lesion in each view of this chart is then measured directly with a planimeter. The total extent of the particular lesion is then taken as the sum of the planimeter readings.

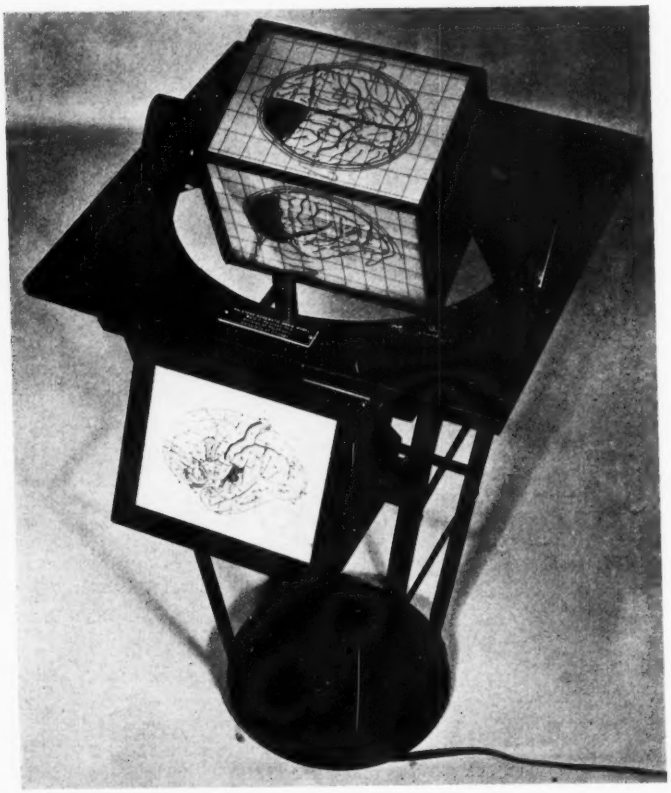

FIG. 2. Schematic brain model for use in expressing the extent of neurosurgical lesions in quantitative terms. See text for detailed description.

It should be noted that this final measurement is a relative measure and not an absolute measure of the surface extent of the lesion. Where the technique is systematically applied, however, the obtained measurement may possibly prove to be even more reliable than post-mortem histological reconstruction, where absent tissue boundaries and shrinkage incident to preparation operate as uncontrollable factors in measurement.

The schematic brain model with its support is portable. It has been found by neurosurgeons to be quite useful in the classroom as a teaching and demonstrational device for students of neurological surgery and for the presentation of neurosurgical histories to medical conferences.

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THE POSTERIOR TIBIAL REFLEX

A REFLEX OF SOME VALUE IN THE LOCALIZATION OF THE PROTRUDED INTERVERTEBRAL DISC IN THE LUMBAR REGION

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Since the recognition of the protruded intervertebral disc as a pathological entity great interest has been shown in clinical localizing signs by neurologist, orthopedist, and neurosurgeon.

For example, it is frequently pointed out that such lesions between the 3rd and 4th lumbar vertebrae may lead to depression of the knee jerk on the affected side and that protrusion between the 5th lumbar vertebra and the sacrum may cause diminution or absence of the ankle jerk. As far as we know there is no reflex commonly used for detection of protrusions between the 4th and 5th lumbar vertebrae.

The knee jerk depends for its activation upon an intact nerve supply to the quadriceps femoris muscle, which is supplied by the 2nd, 3rd and 4th lumbar roots, chiefly the latter two. The ankle jerk depends upon an intact nerve supply from the 1st and 2nd sacral and perhaps 5th lumbar roots. It therefore becomes a matter of choosing a muscle with easily accessible tendon, innervated by a supply arising between the aforementioned two, to obtain a reflex capable of demonstrating a lesion at the L-4 to L-5 interspace. The posterior tibial muscle, innervated by the 4th and 5th lumbar roots, seems a likely choice.

One hundred patients with no complaints referable to the back or lower limbs were tested. In 31 of these we could obtain no posterior tibial reflex on either side. In 28 of the remaining cases the reflex could be obtained only with reinforcement. In 41 there were active reflexes, easily obtainable.

Twelve patients with protruded intervertebral disc at the L-4 to L-5 level were next examined. All had demonstrable defects by myelography. Eight of these had a depressed or absent posterior tibial reflex on the affected side while retaining normal knee and ankle jerks. Five were verified by operation while three remained unexplored.

In four cases we could elicit no posterior tibial reflex on either side. This was not regarded as significant in view of the large number of normals without demonstrable reflexes.

Several patients with a protrusion between the 5th lumbar vertebra and the sacrum have been found with a diminished posterior tibial reflex on the affected side, but never without greater depression or absence of the ipsilateral ankle jerk.

The tendon of the posterior tibial muscle is easily located just behind and above the internal malleolus of the tibia (Fig. 1).

The posterior tibial reflex is, like all tendon reflexes, most easily obtained when the tendon is placed on slight stretch. Since inversion of the foot is our index of reaction, it is necessary to have the foot in neutral position or slight eversion.

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Two positions have proved of value. The more simple (Fig. 2) is as follows: The patient remains prone on the examining table with the feet extending slightly beyond the end. The legs are relaxed. The leg to be examined is flexed slightly at the knee and supported by the examiner.

FIG. 1.

FIG. 2.

The second position (Fig. 3) is of value when both reflexes are present and is used to more clearly demonstrate a diminution on one side. The patient is placed on the side with the leg to

be examined undermost. This leg is then placed over the edge of the table and supported by the examiner. The added power now needed to invert the foot against gravity more effectively demonstrates a difference.

FIG. 3.

SUMMARY

An attempt has been made to isolate a reflex that would be of value in localizing lesions at the interspace between the 4th and 5th lumbar vertebrae.

The posterior tibial reflex has been found useful in this respect.

One hundred patients with no complaints referable to the back or lower limbs were examined as controls. In almost one-third of these no reflex could be elicited. In 40 per cent of the remainder it could be elicited only by reinforcement. The reflex is therefore not ideal. The absence of the posterior tibial reflex on both sides is not significant in a protruded disc suspect. Its sole value as a clinical localizing sign is in those cases where it is diminished or absent on one side only.

Eight patients with protruded disc at the L-4 to L-5 level have been found with normal ankle jerks and knee jerks and an absent or diminished posterior tibial reflex on the affected side. All of these had demonstrable defects by pantopaque myelography. Five have been operated upon and typical herniations removed from each.

CONCLUSIONS

1. A reflex of some value in the localization of protruded discs in the lumbar region has been isolated.
2. The manner of its elicitation has been described.

Gratitude is expressed to Lt. Colonel J. E. Scarff, Lt. Colonel William Cox, Major Neil Rogers, and Major B. B. Whitcomb, on whose wards these patients were seen.

MEETINGS

SCANDINAVIAN NEUROSURGICAL SOCIETY

The next meeting of Nordisk neurokirurgisk Forening will be held in Stockholm, September 13–14, 1946. Members of the Harvey Cushing Society and of the Society of British Neurological Surgeons have been cordially invited to attend.

The Nordisk neurokirurgisk Forening was founded at a meeting in Stockholm on September 22, 1945, and the first scientific session was held in Copenhagen, February 22–23, 1946. Membership is limited to active neurosurgeons in Sweden, Denmark, Norway, Finland and Iceland.

The officers are as follows: Dr. Herbert Olivecrona, Stockholm, President; Dr. Eduard Busch, Copenhagen, Vice-president; Dr. Olof Sjöqvist, Stockholm, Secretary.

HARVEY CUSHING SOCIETY

The 14th annual meeting of the Harvey Cushing Society will be held in Boston, October 10, 11 and 12, 1946. The officers are as follows: Dr. Franc D. Ingraham, Boston, President; Dr. Gilbert C. Anderson, New Orleans, Vice-president; Dr. Louise Eisenhardt, New Haven, Secretary-Treasurer.

CYSTIC CEREBELLAR ARACHNOIDITIS

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Cystic cerebellar arachnoiditis is a pathological state of scarring of the leptomeninges in the posterior cranial fossa, resulting in an internal hydrocephalus and a cystic accumulation of fluid at the site of the cisterna magna.

The condition is not too uncommon in neurosurgical clinics.[1,2,3] However, in post-traumatic states it is rarely considered in the differential diagnosis. The literature was reviewed in 1923 by Horrax[3] at which time some 28 cases had been described.

The probable pathogenesis of the condition is as follows. The patient develops an inflammatory reaction of the leptomeninges in the region of the cisterna magna as a result of bloody, bacterial or contiguous inflammatory reaction present in the subarachnoid space. This causes scarring and thickening of the meninges in this region and results in partial obstruction to the flow of cerebrospinal fluid from the cisterna magna to the remainder of the basal cisterns and subarachnoid spaces over the hemispheres. As a result of this partial obstruction the cisterna magna gradually becomes dilated, resulting in an arachnoidal cyst.

This cyst acts as a space-consuming mass in the posterior fossa, elevates the cerebellar hemispheres and pushes the cerebellar tonsils into the foramen magnum. The partial obstruction of the cerebrospinal fluid flow results in back pressure throughout the ventricular system, with its consequent dilation and the development of an internal hydrocephalus.

The clinical picture in the traumatic type is characterized by symptoms of an expanding lesion in the posterior fossa following cranio-cerebral trauma. The degree of trauma may be very mild, as is demonstrated in the case below. The symptoms may be divided into two groups: those of obstruction to the flow of cerebrospinal fluid with resultant hydrocephalus, and those due to the local effects of pressure of the arachnoidal cyst. The former are the signs of increased intracranial pressure (i.e. headache, papilledema, bradycardia, abducens paralysis, episodes of syncope, transient episodes of blindness, etc.); while the latter are signs of focal pressure on the surrounding structures (i.e. nystagmus, ataxia, partial or complete paresis of the 5th, 7th, or 8th cranial nerves, etc.). The picture, however, is primarily that of a space-occupying mass increasing slowly in size. It cannot be clinically differentiated from the picture produced by a primary neoplasm unless the relationship to trauma or infection arouses suspicion. Skull X-rays and spinal fluid studies are of no value in the differential diagnosis. Lumbar puncture is of

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course contraindicated with signs of increased intracranial pressure and a mass lesion in the posterior fossa. An interesting symptom was present in the following case, namely that of pain in the suboccipital region which was altered by change in position of the head.

CASE REPORT

A 23-year-old veteran, W.K.M., was admitted to the USNH, Newport, Rhode Island on Nov. 16, 1945, complaining of attacks of suboccipital pain, diplopia, weakness, and numbness of the entire body associated with sweating, and transient attacks of blindness.

While serving in the Army with the paratroopers in September 1943, he sustained an injury. He jumped from a training tower. The lines became entangled and the chute did not open. He came rapidly to the ground landing on the right foot, breaking the impact of his fall by rolling forward as he had been taught to do. He does not recall striking his head although he was "dazed" for a second. His leg was fractured. It was put in a cast and he was kept in bed for 1 week and then was allowed to get up and around on crutches. His leg healed without incident.

About November 1943, 60 days following his trauma, the patient began to notice that if he strained or exerted himself he would feel light-headed as if he were going to faint. On Nov. 19, 1943, he was sent to a recuperation hospital where he noted that any flail movement of the arm, as in throwing a ball or swimming, at times precipitated a sudden severe pain in the midline of the neck in the suboccipital region. He also discovered that if he stood up, flexed the neck sharply forward and stood still for about 30 to 60 seconds the pain would disappear. In March 1944, he had for the first time spontaneous onset of pain without any movement or exertion. These attacks occurred at irregular intervals from once a day to once every 4 or 5 days. The pains lasted from 2 to 10 minutes before relief. They were accompanied by a feeling of syncope although he never actually lost consciousness. About April 1944 he began to have occasional diplopia. At the same time he noted that the neck pains radiated up over the vertex, and were felt in the bridge of the nose, and behind the eyeballs. The attacks of pain became more frequent and in May 1944, transient episodes of ataxia developed. At times the ataxia was so severe that he was accused of being intoxicated. It occurred in association with the attacks of neck pain, diplopia or with either one separately. In June the pain radiated down the spine and out to the extremities. There was also a sense of numbness and weakness which would accompany the attacks. The pains at this time lasted about $\frac{1}{2}$ hour. Toward the end of the month profuse sweating was associated with the attacks. This sweating occurred about 2 or 3 times a month, with severe radiating pains over the entire body. On July 28, 1944, he was having attacks twice or more a day with only occasional days free from them. He was hospitalized on Sept. 12, 1944. At that time he had a lumbar puncture, X-rays, and numerous consultations. There was no further change in symptoms and on Oct. 19, 1944, he was discharged from the U. S. Army.

During January 1945 an intermittent internal squint of the right eye developed. He had been wearing a patch over the eye since discharge. The squint occurred from time to time with the attacks of headaches and generalized pain throughout the body. In September 1945 he began to have transient episodes of momentary blindness. The vision faded out rapidly and returned slowly. Macular vision returned first and then the fields cleared up. These attacks occurred 2 to 3 times a week. In October 1945 the first episode of true syncope occurred. Subsequently the patient had repeated episodes of the character of the above, which precipitated his hospitalization.

Examination. Past and family histories were irrelevant. The patient was a nervous healthy-looking white male 23 years of age. Temperature 98.6; pulse 70; respiration 20; blood pressure 120/70. General physical examination was negative. Neurological examination revealed the following positive findings: (1) Bilateral choked discs, 5 D. right, and 4 D. left, with proliferation of scar tissue around the discs suggestive of long-standing increased intracranial pressure; (2) paresis of the right external rectus muscle; (3) dilatation of both pupils (0.6

cm.); (4) facial asymmetry suggestive of a central right facial palsy; (5) hyperactive, deep tendon reflexes on the left, and (6) positive Hoffmann's sign on the left. The remainder of the cranial nerves, sensory and motor systems were negative. Routine blood and urine studies were negative. X-ray studies and chest plate were likewise negative. Visual fields showed generalized peripheral constriction of about 20° in the right eye and 40° in the left, with enlargement of the blind spots.

Electroencephalographic report, Dec. 1, 1946: The occipital areas show a rather irregular subdominant alpha pattern at 9–9½ sec ranging from 50–100 μV with poor amplitude regulation. This is broken up by considerable slower activity in the form of isolated 2–5/sec 100 μV waves and persistent base line shift of still slower frequency. Centrals and frontals show some alpha, but are interrupted by moderate amounts of 12–20/sec activity appearing in rough bursts reaching nearly 75 μV on frontals. There are also short rough bursts and scattered waves at 6–8/sec 50–75 μV that may also involve the occipital areas. Despite poor bilateral symmetry neither side is consistently more abnormal. There is a moderate hyperventilation response. An *Abnormal Record* with considerable non-focal slow and fast activity S1(F1).

Subsequent examination on Dec. 10, 1945 revealed an increase in the papilledema in the left fundus to 6 D. while the right remained at 5 D. The papilledema was out of proportion to his other symptoms. Except for complaints of occasional attacks of suboccipital pain, diplopia, and transient episodes of amblyopia he was symptom free. On Dec. 13, 1945 the patient had severe suboccipital pain, complained of feeling numb all over, broke out into a cold sweat and lost consciousness, falling to the floor. He immediately regained consciousness. Pulse was 52; blood pressure 100/50; papilledema had increased to 10 D. on left and remained 5 D. on right. He rallied in several minutes although he was left with a generalized feeling of weakness and profound sweating. There was no other change in his neurological status.

Operation. He was taken to the operating room the same day, where bilateral parietal trephines were done to rule out an atypical subdural hematoma. These revealed just a drop of cerebrospinal fluid in the subarachnoid space and a tight non-pulsating cortex. Bilateral occipital trephinations were made immediately and a ventricular needle was inserted into the left lateral ventricle; 165 cc. of ventricular fluid were replaced with a like amount of air. The X-rays showed symmetrical dilatation of the lateral, 3rd and 4th ventricles with apparent obstruction at the exit of the 4th ventricle (Fig. 1). The aqueduct and 4th ventricle were angulated rostrally.

The patient was then anesthetized with intratracheal ether, placed in the supine position with his head in the cerebellar head rest. A midline incision was made from 2 cm. above the occipital protuberance to the C-6 spine. The muscles were separated in the midline from the spines with the electrocautery. The occipital muscles were dissected from the occipital bone subperiosteally. A routine trephination was made in the occipital bone and the bone was removed with a rongeur along with the arch of the atlas. The dura over the right cerebellar hemisphere was bluer than normal. It was penetrated with a #26 needle and clear colorless fluid was aspirated, which on examination proved to be the same as ventricular fluid. On opening the dura in a T-shaped manner a cyst was seen, which occupied the lower right half of the posterior fossa and bulged into the foramen magnum (Fig. 2). It measured approximately 5 × 4 × 4 cm. There was elevation of the right cerebellar hemisphere and flattening of the cerebellar tonsils, which were pushed anteriorly and down into the foramen magnum against the medulla. The dorsal wall of the cyst was resected and the cerebellar tonsils were then dissected from the surface of the medulla. The cyst was then resected around the side of the posterior fossa toward the cerebellopontine angle. The ventricular system was filled with air and air was seen to escape from the 4th ventricle. The lower part of the right cerebellar tonsil which was deepest in its herniation was resected. The dura was closed and the wound closed in layers with interrupted cotton sutures.

Postoperative Course. The patient did very well. The papilledema began to recede almost

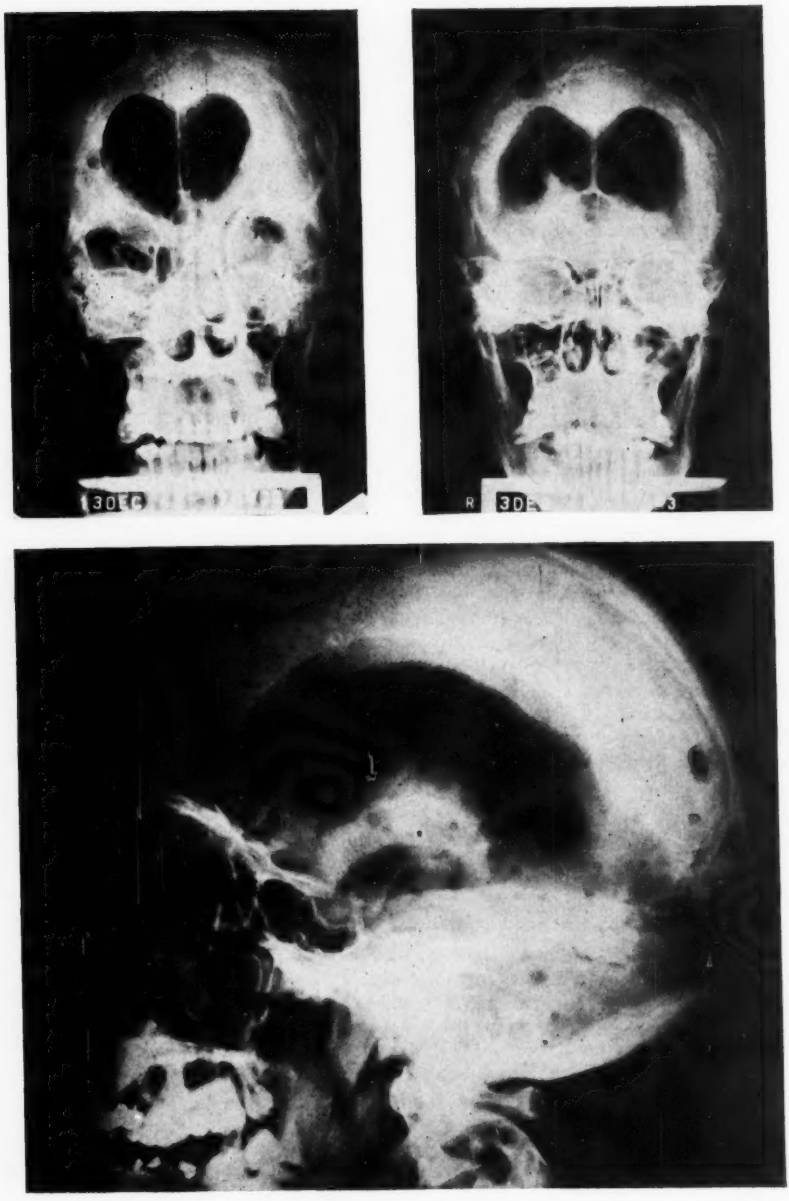

FIG. 1. Ventriculograms showing dilated ventricular system and distorted aqueduct of Sylvius.

immediately and all symptoms except his rectus paresis were gone. During the course of the next 2 weeks the diplopia and rectus paresis disappeared and he was asymptomatic.

Pathological Report. The cyst wall consisted of thin fibrous tissue showing invasion of small round cells plus an occasional polymorphonuclear leukocyte and eosinophile. Impression: Chronic Arachnoiditis.

Subsequent Course. Visual fields on Jan. 25, 1946 showed a complete return to normal.

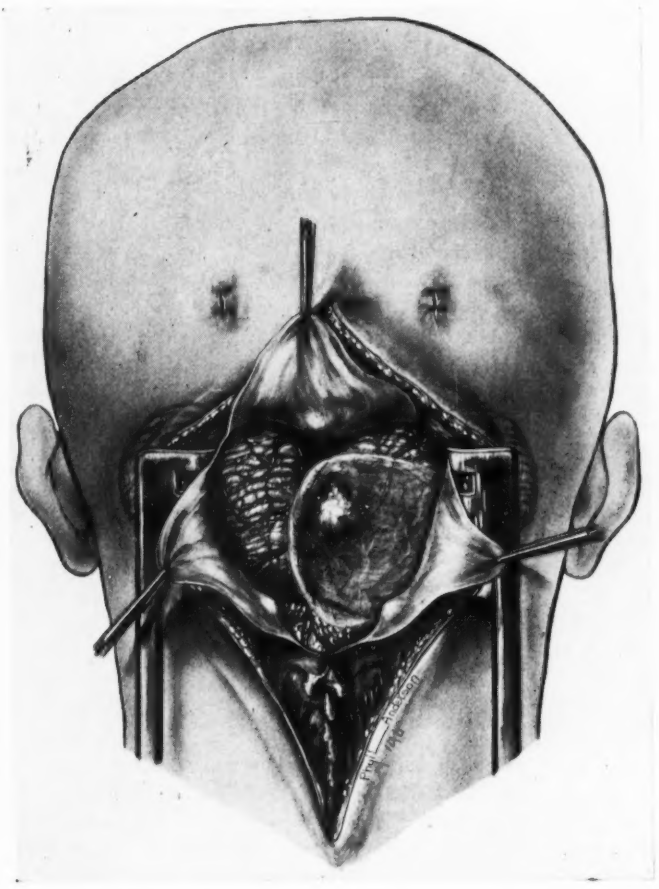

FIG. 2. Arachnoidal cyst as seen at operation.

Psychometric evaluation by the Wechsler-Bellevue and Rorschach methods revealed a high average intelligence with an I.Q. of 111 and no evidence of gross organic deterioration.

Electroencephalographic report, Jan. 29, 1946: The occipital areas show a moderately regular, mostly dominant alpha pattern at about $9\frac{1}{2}$/sec 59 μV with some surging to 100 μV. The base line drift has cleared up completely as has the scattered but common 2–4/sec activity, leaving only rare isolated 5–6/sec 50 μV waves from either side. There is a fairly

good spread of alpha into central and frontal areas, with only rare slowing. The surging 12–20/sec pattern has almost completely disappeared, leaving only rare mild hypersynchrony at about 8/sec. There is a mild hyperventilation response. Despite some residual instability feel record is *probably within normal limits*. Slow activity has almost completely disappeared $9\frac{1}{2}$/sec.

Neurological examination on Feb. 1, 1946 showed that the papilledema had completely subsided and that the patient was normal in all respects. On follow up at the end of 6 months the patient remained symptom free and neurologically negative.

DISCUSSION

This case is of interest in several respects. It demonstrates how minimal cranio-cerebral trauma may produce serious sequellae which mock in many ways a post-traumatic cerebral syndrome. The diagnosis could not be established without the onset of papilledema or air studies. In retrospect the condition probably could have been suspected earlier if the progression of the patient's neurological signs had been noticed.

Following his injury, there was a period of 2 months when there were no symptoms or neurological signs. During this latent period scarring of the meninges was taking place. The first symptom was a feeling of lightheadedness which probably represented the first partial obstruction of cerebrospinal fluid causing a transient rise in intraventricular pressure and the subsequent symptoms. He developed next suboccipital pain on exertion, which was relieved by flexion of the neck. This was undoubtedly due to protrusion of the beginning cyst into the foramen magnum with pressure upon the dorsal primary divisions of the upper cervical nerves. The sharp flexion of the head would then tend to pull the cyst out of the foramen with prompt relief of pressure on the nerves and consequent relief of pain. The size of the cyst increased and, therefore, the patient began to develop spontaneous onset of pain. At this time the symptoms of acute hydrocephalus had their onset with a feeling of syncope associated with headaches which became generalized, spreading from occiput to vertex. The 6th nerve on the right then became progressively involved. The mechanism of this is not entirely clear. It might have been on the basis of acute increased pressure due to acute hydrocephalus, but this probably was not the case, since the attacks would not always be accompanied by generalized headaches, syncope, etc. In looking for another explanation the following was noted. The cyst was predominantly in the right posterior fossa and it is not too untenable to conceive that sudden swelling of the cyst would produce elevation of the cerebellar hemisphere and compression or traction of the right 6th nerve in its relation to the tentorium. The fact that signs of cerebellar compression began about the same time tend to substantiate this conclusion. Acute pressure of the cystic mass was most certainly directed on the nuclei of the dorsal columns and their tracts, possibly accounting for the radiating numbness and paresthesiae. The patient continued to have attacks of acute intermittent hydrocephalus, which slowly dilated the ventricular system.

Attacks of sudden sharp increase in intracranial pressure began next to

affect the visual system, and papilledema resulted. This made itself manifest to the patient by attacks of amblyopia. The attacks of syncope were another manifestation of acute hydrocephalus.

The intermittency of symptoms and the complete freedom of symptoms between attacks are explained by the periodic filling and emptying of the cyst; as changes in posture of the head, exertion and other factors altered the spatial relationship of the posterior fossa structures. The relief obtained by acute flexion of the head, pulling the cyst and cerebellar tonsils from the posterior fossa, is worthy of note as a symptom.

Examination of the fundi revealed a choked disc of long standing, which would be expected from his history. The sudden rapid increase in papilledema undoubtedly pointed to the fact that the hydrocephalus could no longer be considered intermittent and that the case became one of surgical emergency.

The case is of interest in that all neurosurgeons have seen patients with suboccipital neuralgia following trauma similar to that present early in this case, which was suddenly and permanently relieved by lumbar puncture. It is quite probable that such an obstructing membrane as in the above case, in the early stages, is ruptured by withdrawal of fluid allowing normal circulation of cerebrospinal fluid with subsidence of symptoms.

SUMMARY

1. The probable mechanism of cerebellar cystic arachnoiditis has been briefly outlined.

2. The probable pathogenesis of symptoms has been described and discussed.

3. An illustrative case of the disorder has been described in detail.

4. The possible relationship of this condition to post-traumatic suboccipital neuralgia has been pointed out.

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PAIN ON STIMULATING THE DISTAL SEGMENT OF DIVIDED PERIPHERAL NERVES

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(Received for publication July 15, 1946)

DURING neurorrhaphy under local anesthesia it was observed that stimuli applied to the *distal* end of a severed peripheral nerve frequently caused pain. It was also found that the distribution of pain so induced could be shifted by blocking an adjacent intact nerve. The theoretical and practical implications of these observations led the authors to submit the following case reports.

CASE REPORTS

Case 1. Median Nerve. Arm: lower third.

J.F.D., aged 36, sustained a gunshot wound of the lower third of the left arm in March 1945. During debridement it was noted that the median nerve was completely severed. There had been no recovery of median nerve function, either sensory or motor, since the injury.

On Sept. 5, 1945, approximately 6 months later, exploration revealed a gap between the two ends of the divided nerve which was too great to permit immediate repair. The proximal and distal segments were therefore dissected free of the surrounding tissues and thus mobilized so that a temporary end-to-end "bulb" suture could be done, in order to stretch the nerve sufficiently for subsequent anastomosis.

On Oct. 24, 1945, 7 weeks after the first-stage procedure, the median nerve was again exposed, under local infiltration of the skin and the proximal or central stump of the nerve. The proximal stump was then cleanly cut at the junction of the neuroma and normal-looking nerve. No pain was experienced during this part of the procedure.

The distal segment of the nerve was now cut in like fashion, no anesthetic agent having been applied to it. On making this distal cut, a sensation of pain was felt in the mid-palmar area. Faradic stimulation of the distal stump was then carried out, which elicited a burning type of pain in the mid-palm regardless of whether the bipolar electrodes were placed on the cut section of the nerve or on its surface. (A Hinsey-Geoghan stimulator was used at a current strength of 1.0 to 1.5 volts; 60 cycles; with the bipolar electrodes 3 mm. apart.)

Mechanical and electrical stimulation of the adjacent tissues did not elicit pain in this case. No motor response accompanied these tests. The distal segment of nerve was carefully walled off with dry gauze pads for a distance of 5 cm. to prevent "spread" of electrical current. (By mechanical stimuli is meant compression of the nerve with delicate bayonet forceps, which elicited responses similar to electrical stimulation.)

The ulnar nerve was now blocked with 2 per cent procain just above the olecranon groove, and the distal end of the median nerve again stimulated. This time the patient complained of pain over the dorsal surface of the 2nd and 3rd fingers instead of the palm.

Since the median nerve was completely severed, the only central connections were the ulnar and radial nerves. The ulnar nerve evidently participated in the relay of impulses responsible for a feeling of pain in the palm, for when it was blocked the pain was then referred to the dorsal aspect of the 2nd and 3rd fingers. The sensation of pain must then have been a function of the sole remaining nerve to the hand: the radial.

Microscopic examination of cross sections of the proximal and distal segments of the sev-

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ered nerve (myelin and Bodian stains) in this and the following cases, taken from the site of the proposed suture, showed relatively normal nerve fibres in the proximal specimen, but no intact fibres in the distal section. There must, however, have been some functioning fibres in the distal segment capable of conducting impulses from the point of stimulation to the periphery.

Case 2. Median Nerve. Forearm: lower third.

P.F. was wounded on April 16, 1945 by an exploding grenade. Examination 7 months later revealed "complete paralysis of the sensory component of the median nerve from the wrist down." At operation on Nov. 16, 1945, extensive scarring was found in the lower forearm and wrist. The proximal stump of the divided median nerve ended blindly in scar tissue 5 cm. above the wrist. The distal stump was found 2 cm. below the carpal ligament. It was necessary to do a first-stage "bulb" suture. To accomplish this, the defect of 7 cm. had to be made up by mobilizing the median nerve along the entire length of the forearm.

Examination on Mar. 25, 1945, approximately 4 months later, showed loss of pinprick, heat and pinch perception over the entire volar aspect of the 2nd and 3rd fingers and the distal half of the thumb. A healing cigarette burn was noted on the 3rd finger, but the patient had not been aware of any sensation at the time of burning. On stroking the skin with cotton, dysesthesias could be induced over the volar aspect of the thumb, index finger and thenar eminence, but none over the 3rd finger. All forms of sensation were readily appreciated over the dorsal aspect of these fingers within the radial nerve distribution.

On Mar. 26, 1945 the second-stage repair was carried out under local anesthesia only. The previous first-stage "bulb" suture and neuromata were resected following 2 per cent procain block of the proximal segment of the median nerve. When this had been done, bipolar electrodes were applied to the distal end of the median nerve for electrical stimulation, with the same technique described for Case 1. Pain was elicited over the dorsal aspect of the web space between the thumb and the index finger.

The superficial branch of the radial nerve was now blocked with 2 per cent procain above the level of the wrist, and the distal end of the median nerve stimulated again with the same current. No pain could be elicited on repeated trials. Evidently the radial nerve was responsible in this case for the central relay of impulses induced by stimulation of the median nerve stump.

Case 3. Ulnar Nerve. Forearm: distal third.

J.A., aged 27, had been wounded in action in February 1945. There had been complete paralysis of all ulnar nerve function below the level of the injury due to complete severance of the nerve in the lower third of the forearm. In August 1945 a first-stage attempt to repair the nerve was performed, which required extensive mobilization of both segments of the nerve before an end-to-end "bulb" suture could be accomplished, so great was the initial defect.

On Oct. 17, 1945, approximately 8 months after injury, and 2 months after the first-stage procedure, neurorrhaphy was carried out under local anesthesia. A large fusiform swelling marked the site of the "bulb" suture. The nerve above and below this was so badly scarred that an additional 10 cm. had to be resected. Satisfactory anastomosis was eventually achieved after full mobilization of the nerve and flexion of the forearm and wrist. The dorsal cutaneous branch of the ulnar nerve was dissected free as far as the dorsal aspect of the wrist, and one of its three subdivisions at this point sacrificed in order to release tension. As this branch was cut the patient cried out with pain, which he referred to the hypothenar area. Electrical stimulation of the distal end of the divided ulnar nerve itself was then carried out. This resulted in pain referred to the *median* nerve distribution of thumb, index and middle fingers. In this case, the effects of stimulating the isolated stump of the ulnar nerve were evidently relayed in a central direction by the median nerve.

Case 4. Ulnar Nerve. Forearm: middle third.

J.J. received a severe gutter type wound in the medial aspect of the left forearm in February 1945, which resulted in a considerable loss of substance of the flexor carpi ulnaris muscle and overlying skin, and complete enduring paralysis of all ulnar nerve function below the level of injury.

On Dec. 3, 1945, under local cutaneous anesthesia supplemented by 2 per cent procain block of the proximal stump of the ulnar nerve, the latter was exposed from the middle third of the arm to the wrist. The nerve was found to be completely severed and embedded in dense scar tissue at the middle third of the forearm. The ulnar artery was thrombosed and was therefore resected at this point between ligatures. The distal end of the ulnar nerve was dissected down to the wrist and its dorsal cutaneous branch freed as far as the dorsal aspect of the hand.

Using bipolar electrodes, the isolated portion of the ulnar nerve was electrically stimulated with the technique described for Case 1. This caused pain over the median nerve distribution of the 3rd and 4th fingers, as did mechanical stimulation.

Stimulation of the isolated segment of the ulnar nerve was then repeated after blocking the median nerve in the mid-forearm with 2 per cent procain. Pain was now referred to the radial nerve distribution over the dorsum of the hand at the base of the 3rd and 4th fingers. Stimulation of the dorsal cutaneous branch of the ulnar nerve also elicited pain in this distribution.

As a check on the effectiveness of the median nerve block, the median nerve was now stimulated electrically above or central to the block; that is, at the level of the antecubital fossa. This resulted in contraction of the muscles supplied by the median nerve above the block, and pain referred to the entire median nerve distribution of the hand.

It appears, therefore, that prior to median nerve block, the effects of stimulating the distal stump of the divided ulnar nerve were relayed centrally by the median nerve; but that after the latter was blocked, by the radial nerve.

DISCUSSION

Observations similar to those just described were noted during the course of other operations (approximately one in five) carried out under local anesthesia for the repair of median and ulnar nerves that had been completely severed in the arm or the forearm. Other neurosurgeons have remarked upon similar findings.[3,4] Unfortunately exact statistics cannot be supplied since no records of this phenomenon were kept until the latter months of the War. Nor was this type of observation possible during repair of nerves in the lower extremities, since spinal or general instead of local anesthesia was used.

Stimulation or dissection of scar tissue adjacent to severed nerves in some instances led to intensely sharp pain, localized to the operative site, and occurring almost immediately after stimulation was applied. In contradistinction to this, the pain evoked by stimulation or dissection of a distal nerve stump (1) was referred peripherally and not locally; (2) was burning in character rather than lancinating or shocking; and (3) occurred after an appreciable lag following stimulation.

It is noteworthy that in most cases no pain could be elicited by any form of stimulus applied directly to the fibromatous enlargement or "distal neuroma" of the isolated segment of nerve, although when the same stimulus was applied to the nerve itself immediately below or distal to this enlargement, the nerve proved to be exquisitely sensitive. In fact, there appeared to be a zone, usually 1 cm. in length, having a markedly decreased threshold as com-

pared with more distal portions of the nerve. This "trigger zone" was apparently similar to the local area of diminished threshold to stimuli applied at the boundary between scar and the distal stump of transected spinal cords in paraplegia.[7] The explanation of this finding remains for the moment obscure.

In Case 4, after median nerve block in the mid-forearm, the brachial artery was stimulated electrically in the antecubital fossa, with consequent pain of considerable severity referred to the entire median nerve distribution. It is therefore concluded that, in this case at least, there were neural communications between the artery and the median nerve above the level of the forearm. Corroborating this view is the fact that in several cases explored by us and others at the same hospital, distinct nerve branches were demonstrable coursing between the median nerve in the mid-portion of the arm and the adjacent brachial artery. Thus, while the main autonomic supply of the arteries of the upper extremity may be derived from the median nerve in the forearm,[10] there also appear to be significant connections in the upper portion of the arm as well.

Stimulation of the divided ulnar artery in two patients, each having long-standing severance of the adjacent ulnar nerve, resulted in pain referred to the volar carpal region and hypothenar eminence, upon stimulation of the distal end of the artery, but no sensation upon stimulation of the proximal end. (No tests with intra-arterial injections were carried out.[5])

At this point it should be emphasized: (1) that every precaution was taken so that no spread of electrical current should occur; (2) that no motor response followed stimulation of the distal nerve segments described; and (3) that the area supplied by each severed nerve described was totally insensitive to all forms of cutaneous stimulation as tested clinically prior to neurorrhaphy, with the possible exception of Case 2.

From these and similar cases it is clear therefore that stimulation of the distal, chronically isolated segment of a completely divided nerve "A," may result in pain referred to the area innervated by an adjacent *intact* nerve "B." It is also evident that after blocking nerve "B," the pain may then be referred to a zone supplied by another intact nerve "C," which in turn apparently serves as the central relay for sensory impulses induced by stimulation of nerve stump "A."

Sensory impulses induced by stimulation of an isolated nerve segment may reach an intact nerve by direct anastomosing branches similar to those motor branches described in the illuminating communication by Murphey *et al.*[6] Stimulation may also induce changes in the chemical mediator[2] within peripheral sensory units where terminal twigs of adjacent nerves overlap and arborize.[1] It is also possible that the painful sensations described may be due to impulses induced in the peripheral network of the autonomic system, thus evoking a local sympathetic reflex,[8] the effects of which in turn are relayed centrally by an intact peripheral nerve subserving that area. The pain elicited by stimulation of long-divided distal nerve stumps (prior to their

operative section) can hardly be attributed to an "axonal reflex" effect[9] since the latter depends upon freshly cut nerve.

The nature of the fibres presumed to be functioning in the distal segment of chronically divided nerves must remain for the moment a matter of speculation. Several possibilities, however, come to mind: (1) Some fibres may have preserved their viability without undergoing degeneration, by virtue of continuity with viable fibres in adjacent intact nerves. This is in keeping with the fact that anastomotic branches frequently exist between the ulnar and median nerve.[6] (2) Regeneration of fibres across the gap of scar tissue separating the two ends of a long-divided nerve is possible but unlikely, since nerve fibres could not be demonstrated in specimens of the scar, and since there was no clinical evidence that any such regeneration had occurred. Moreover, if regenerating nerve elements had bridged the gap, they would presumably have entered the distal nerve segment by way of its severed end, marked by fibroma formation. However, stimulation of the distal fibroma did not result in pain, while stimulation of the nerve below it did. It seems unlikely therefore that nerve fibres responsible for pain in the distal segments of these divided nerves had traversed the gap between nerve ends. The only alternative is retrograde regeneration of fibres from the anastomotic branches described by Murphey *et al.*[6] after careful studies of motor function of these same nerves, or retrograde regeneration of fibres of the autonomic nervous system, in accordance with their well recognized proclivity for regeneration.[10]

The immediate practical significance of these observations to the neurosurgeon has been illustrated by several cases we have had to re-explore in order to resect a neuroma-in-continuity and carry out neurorrhaphy. This procedure had not been done at the initial operation because pain had been elicited on stimulating the distal segment of the nerve. Accordingly the neuroma was not resected initially as it should have been. Pain on stimulation distal to a neuroma cannot be relied upon as an index of functional continuity of that nerve, even though there is no visible break or gap in continuity at the site of injury.

CONCLUSIONS

1. Stimulation of the distal stump of certain long-divided peripheral nerves elicits pain.

2. The pain may be referred either to the sensory distribution of the divided nerve or of an adjacent intact nerve and can be abolished, or its distribution shifted, by blocking an adjacent intact nerve.

3. Central relay of the effects of distal stump stimulation is evidently mediated by one or more intact peripheral nerves closely related to the injured one.

4. The pain described above may be the result of, (a) impulses reaching intact nerves via direct anastomotic branches between the divided and intact nerves; (b) changes induced by stimulation in the chemical mediator of

sensory units at the zone of overlap between closely related peripheral nerves; or (c) stimuli affecting peripheral fibres of the autonomic nervous system.

5. The practical significance of these observations is discussed.

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SPINAL EXTRADURAL CYSTS, CONGENITAL AND ACQUIRED

REPORT OF CASES

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CASES of congenital and acquired extradural cysts are reported together in this paper with the purpose of comparing them from etiologic and pathologic standpoints.

I. CONGENITAL SPINAL EXTRADURAL CYSTS

Congenital extradural cysts are rare. The last report of a case in the literature was that of Good, Adson and Abbott (1944).[8] In 1934 Elsberg, Dyke and Brewer[4] reported 4 cases and described the cyst with its attending symptoms and signs as a definite clinical entity. The 4 cases represented the incidence in 250 cases of spinal tumor. Adelstein (1941)[1] found only 16 cases reported in the literature and added one of his own. He found that only 3 cases had been reported previous to 1934: one by Schlesinger in 1898,[13] one by Krauss in 1908,[10] and one by Mixter in 1932.[12] In addition to those listed by Adelstein there are 4 cases in the literature as follows: one reported by Turnbull in 1939;[14] one by Mayfield and Grantham in 1942;[11] one by Meredith quoted by Mayfield and Grantham;[11] and one by Good, Adson and Abbott.[8] Our case is apparently the 22nd to be recorded.

The Clinical Syndrome. All of the cases that have been reported are strikingly similar. The lesion manifested itself and was discovered in most instances before the patient was 20 years of age. The cyst is almost consistently located in the mid-thoracic region. Since it is an extradural expanding mass situated for the most part posterior to the cord, it compresses the latter forward and provokes symptoms and signs of such compression: motor weakness in the lower extremities associated with spasticity, hyperactive deep reflexes and the Babinski sign; varying degrees of loss of sensibility to touch, pain and temperature discrimination; some loss of sense of position and two-point discrimination, and in some cases a loss of bowel and bladder control.

A spinal puncture below the lesion reveals a marked increase in protein in the spinal fluid and a partial or complete block when the Queckenstedt test is performed.

Roentgenogram. The roentgenogram of the dorsal spine is of special interest because it has consistently revealed a kyphosis or scoliosis. Cloward and Bucy[2] pointed out this association of kyphosis with the cyst and believed the deformity to be due to impaired venous drainage from the neighboring vertebral bodies. In addition to the kyphosis there is usually a widening of

the spinal canal due to a smooth erosion of the pedicles over several segments.

Thus when the symptoms and signs of a compressive intraspinal lesion are presented in an adolescent patient and these are associated with the type of deformity of the dorsal spine described, one may reasonably suspect an extradural cyst.

CASE REPORTS

Case 1. #68945. F.M., a Mexican male, aged 13, was admitted to the Colorado General Hospital, Denver, Colorado, on April 30, 1945. About 8 months before, he felt that his legs were getting weak. He fell occasionally because of the weakness. He then gradually lost sensation in the legs. Numbness began in the feet and gradually ascended to the abdomen. A severe burn on the skin caused no pain. Lately he had experienced difficulty in starting the urinary stream but still retained adequate control of bowel and bladder function. Lately he had complained of a painful girdle-like band about his waist just above the level of the umbilicus.

Examination. He was well developed and well nourished. The general and neurological examination gave essentially negative findings except for the following: He could barely walk with aid and the gait was markedly ataxic. The Romberg test was markedly positive. The patellar and Achilles tendon reflexes were hyperactive. There was sustained clonus of the ankles on both sides. The abdominal reflexes were absent; cremasteric reflexes present. The Babinski sign was present on both sides. There was a marked loss of all skin sensibilities up to a rather well defined level at about D-8. The usual tests indicated very poor coordination of the lower extremities and alternate motor movement was poorly performed. He had no sense of position of the large toes.

Laboratory Tests. The urine was normal. Blood studies revealed 12.9 gm. of hemoglobin, 9,400,000 erythrocytes, and 8,400 leukocytes of which 58 per cent were polymorphonuclears and 36 per cent were lymphocytes. There was a marked hypochromia and anisocytosis and because of this a biopsy of the marrow of the sternum was taken. It revealed no unusual diagnostic pattern. The blood serology was negative.

Spinal Puncture. Fluid pressure was 90 mm. of water in the prone position. The Queckenstedt test demonstrated a complete spinal block. The protein content of the spinal fluid was 140 mgm. per cent; sugar, 54; Wassermann test, negative.

Roentgenograms. The skull and long bones were negative. The dorsal spine showed a mild scoliosis with convexity to the right. The center of the convexity was at D-5 and 6. The spinal canal was widened and the pedicles were narrowed at D-6, 7 and 8.

Diagnosis. Intraspinal tumor, probably extramedullary and located at about D-7 and 8.

Operation. On May 18, 1945, under avertin anesthesia, the laminal arches of D-7 and 8 were removed. A structure that was taken for dura mater came into view, and it appeared to have the natural color and texture of dura. It was under tension and was incised in the customary manner. Clear transparent fluid gushed forth leaving a hollow cavity with no visible spinal cord. This was at first confusing. A blunt instrument was passed upward and downward within the cavity and met with obstruction at the lower limit of D-4 and the upper limit of D-10. The laminal arches of D-4 to D-10 were removed. It then became obvious that the lesion consisted of a fusiform cyst with pads of fat at both poles (Fig. 1). It was situated in the midline, extradural and posterior to the dura. It was of such a size in its maximum diameter that it could have filled a normal spinal canal, and it severely compressed the dura and spinal cord forward. The cyst was removed without difficulty, having to be severed from the dura at only one attachment the size of a pinhead and close to the lower pole. It was clearly established that there was no fistula or communication with the subarachnoid space. The dura was opened and except for the marked compression of the cord there was no other abnormality of the intradural contents noted. The spinal canal was wider than normal at D-6, 7 and 8 in keeping with the X-ray findings, and the severe compression of the nerve roots intraspinally could easily have accounted for the patient's complaint of girdle pain. The wound was closed in the usual manner.

Postoperative Course. This was uneventful. At the time he was discharged, June 25, 1945, he had already made considerable recovery of motor power and sensation in the lower extremities.

Pathology. The cyst proved to be an intact completely closed structure, fusiform in contour and presenting an abundance of fat at each end. Both the inner and outer aspects of the wall were strongly reminiscent of dura in respect to color, thickness and texture.

Microscopically the wall was well differentiated into layers that simulated arachnoid, dura and an external well differentiated vascular layer (Fig. 2).

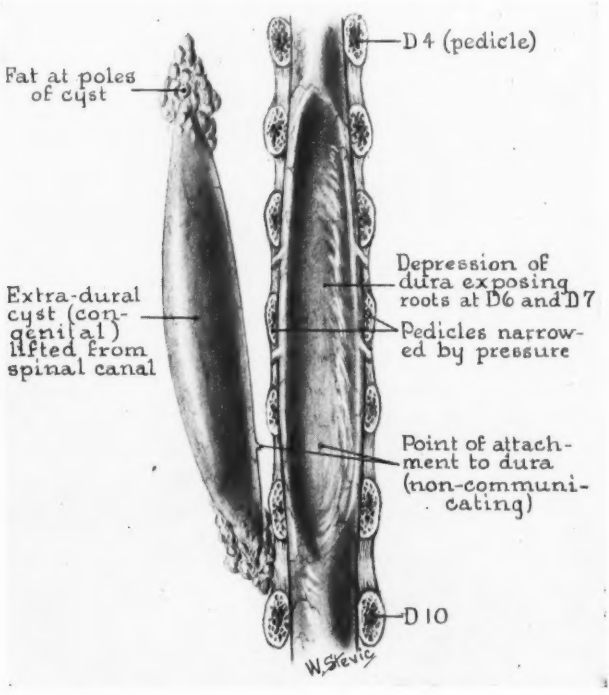
D 4 (pedicle)
Fat at poles of cyst
Depression of dura exposing roots at D6 and D7
Extra-dural cyst (congenital) lifted from spinal canal
Pedicles narrowed by pressure
Point of attachment to dura (non-communicating)
D 10
W. Stevig

FIG. 1. Case 1. Drawing of the congenital extradural cyst.

Discussion. Our case presented the usual features of the syndrome of congenital extradural cyst except possibly in respect to girdle pain, which was not a definite symptom in the other cases that have been reported. When the cyst is large and compresses the neighboring spinal nerve roots, as in our patient, girdle pain is a natural consequence.

In respect to etiology, authors have generally subscribed to the theory that the cyst is a congenital diverticulum of the dura.[1] The fact that in some cases the cyst communicated with the subarachnoid space led to the hypothesis that the former resulted from a herniation of the arachnoid through a defect in the dura. Our case presented no weakness or defect in the dura,

and after studying the histology of the cyst wall one of us (O. H.) is of the opinion that these congenital cysts arise from misplaced or detached rests of cells in a manner similar to the development of ganglia about the wrist and epithelial inclusion cysts.

If there is a communicating channel of fair size between the subarachnoid space and the cyst, the pressure in the latter would probably not be as great as that in a closed or non-communicating cyst. The cyst reported by Good,

FIG. 2. Case 1. Photomicrograph of cross section of congenital extradural cyst (×115). Note high degree of organization and structures that closely simulate arachnoid and dura. There is a well defined vascular layer external to the "dural" layer containing well formed blood vessels. The structure is well differentiated but obviously congenitally aberrant in origin. Compare this well differentiated cyst wall with the acquired "scar tissue" wall in Fig. 5. A, inner lining of cyst wall, probably mesothelial. D, outer heavy covering closely resembling dura. V, well formed blood vessels in vascular layer.

Adson and Abbott[8] communicated with a channel large enough to transmit lipiodol. This may account for the fact that their patient did not develop symptoms and signs until at a much later age than usual.

Differential Diagnosis. There are few lesions that present the combination of symptoms and signs that have characterized the congenital extradural cyst; to wit—its manifestation during youth, the symptoms and signs of an expanding mass in the dorsal spinal canal which compresses the spinal cord and shows a partial or complete block on the Queckenstedt test, and roent-

genological findings which betray an expanding mass of long standing within the spinal canal. Hence Elsberg, Dyke and Brewer[4] felt justified in proposing that such a syndrome identified the cyst as a diagnostic entity.

The roentgenological evidence of an expanding mass within the dorsal spinal canal that causes widening of the canal and narrowing of the pedicles with some degree of kyphoscoliosis, without evidence of an intraosseous destructive lesion would betray a long-standing, usually benign and probably congenital lesion. This would limit the diagnostic possibilities considerably.

It would not be impossible for the syndrome to be simulated in all of its features by syringomyelia. Kyphoscoliosis is commonly associated with syringomyelia. It is also conceivable that local enlargement of the spinal canal could be caused by a slow expansion of the spinal cord due to an intramedullary accumulation of fluid, although we have never encountered such a case.

Intramedullary tumors may rarely grow to such large proportions and slowly enough to cause the findings under discussion. Almost as if to teach us that complete clinical entities are rare, we encountered such a case of intramedullary tumor (spongioblastoma) just 10 months after studying the case of congenital cyst reported here.

Since the syndrome in all of its features so closely simulated that of congenital extradural cyst and since this type of tumor itself is rare and interesting, we are including the report in some detail.

The rarity of this neoplasm is indicated in the literature briefly as follows: Ford,[7] in analyzing Dandy's series of cord tumors, found only one questionable spongioblastoma in a child of 14. In Elsberg's[3] series of 253 spinal neoplasms there were only 26 primary gliomas of the cord and there were no spongioblastomas in this group. However, 6 cases were cited in the literature: two by Kernohan,[9] three by Elvidge, Penfield and Cone,[5] and one by Foerster and Bailey.[6]

Case 2. Intramedullary Spongioblastoma Simulating Extradural Cyst. W.M., a Mexican male, aged 18, was admitted to the Colorado General Hospital, on Feb. 2, 1946. About $1\frac{1}{2}$ years before admission the patient felt that his legs were getting weak and he did not sense pain when he pinched the skin of his legs. Six months later his mother noted scoliosis of the dorsal spine. On Nov. 26, 1945, because of the weakness of the lower extremities and the "crooked" spine, he was taken to the Spears Sanitarium, a chiropractic institution in Denver, where he was given chiropractic treatments and colonic irrigations. After about 6 weeks in the sanitarium the patient became almost completely paralyzed below the level of D-8. After another 3 weeks he was discharged from the sanitarium and admitted to the Colorado General Hospital.

Examination. He presented a complete loss to all modalities of sensation below D-8 and a partial loss up to the level of D-3. All motor function was paralyzed below the waist except for the fact that he could wiggle both large toes very slightly. There was complete loss of bowel and bladder control.

A spinal puncture at the 3rd lumbar interspace revealed a complete block on the Queckenstedt test and a high protein content in the spinal fluid.

Roentgenogram. The dorsal spine showed a moderate scoliosis to the right. The spinal canal

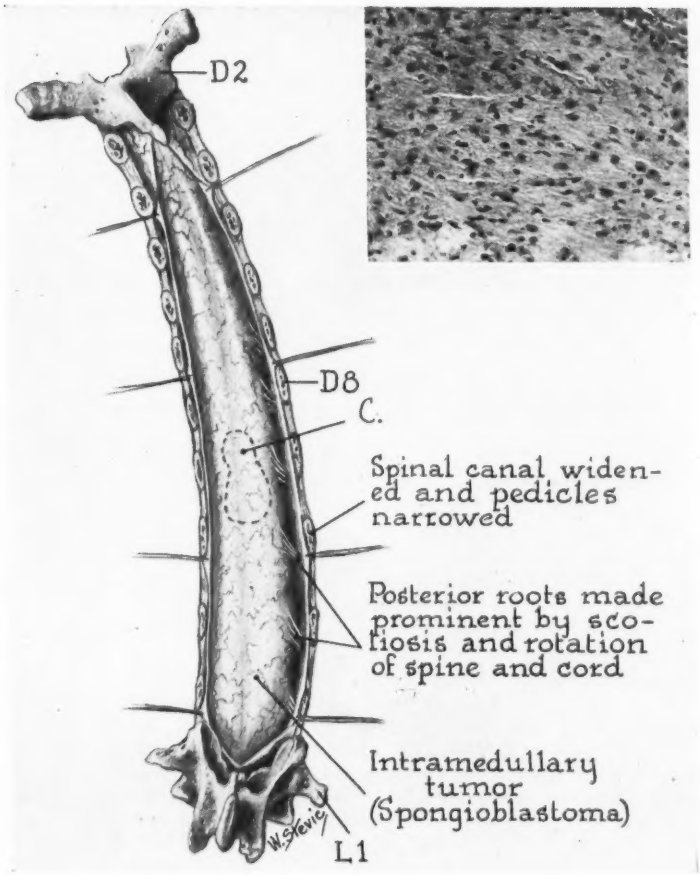

FIG. 3. Case 2. Drawing of the intramedullary neoplasm, which enlarged the spinal cord uniformly from D-3 to below L-1. The pia remained intact everywhere. The slow growth of the tumor resulted in widening of the spinal canal, narrowing of the pedicles, and scoliosis, so that the syndrome closely simulated that of congenital extradural cyst. C, intramedullary cyst containing chocolate-colored fluid. Inset, photomicrograph of the neoplasm (hematoxylin and eosin stain).

was enlarged and the pedicles narrowed from D-5 to D-12. In a lateral view there was a smooth pressure erosion of the bodies of the vertebrae from D-8 to D-12.

Diagnosis. Since we were dealing with an intraspinal expanding mass in a boy of 18 extending over a number of segments and of long enough standing to cause scoliosis and a widening of the spinal canal, the diagnosis was naturally that of congenital extradural cyst.

Operation. Ultimately the laminal arches from D-3 to D-12 inclusive were removed. The spinal cord was uniformly enlarged throughout the entire exposure, being 1 inch in diameter in the mid-dorsal region (Fig. 3). The pia was everywhere intact. An incision was made in the posterior median fissure. The tissue was uniformly brown and there was nothing that grossly resembled cord tissue. A cyst containing chocolate-colored fluid was present (Fig. 3, C). A biopsy was made.

Pathology. Microscopical study revealed the cellular characteristics of spongioblastoma with evidence of old hemorrhage (Fig. 3, inset).

Course. The patient recovered from the operative procedure and although the cord appeared to have been entirely destroyed by neoplasm, he could still voluntarily move both large toes as he did before operation.

Although spongioblastoma is an infiltrating glioma, it usually has a long life history. If it occurs in the brain stem, it usually enlarges the latter uniformly and while tumor cells infiltrate the tissue everywhere, they appear to do so without much destruction of the nerve cells and fiber tracts until late in its history.

II. ACQUIRED EXTRADURAL CYSTS

We have been unable to find a report of this lesion in the literature. The two cases reported here are illustrative and are the only cases of their kind we have encountered.

CASE REPORTS

Case 3. #60779. B.M., a white girl, aged 16, was admitted to the Colorado General Hospital, on May 4, 1943. She complained of pain radiating down both legs of about 10 weeks duration.

On examination she presented an absent Achilles reflex on the right and hypalgesia on the lateral aspect of the left leg. Blood and urine were negative.

Spinal Fluid. The Queckenstedt test gave a normal rise and fall,* but when the pressure in the spinal subarachnoid space was increased, the pain radiating down the lower extremities was aggravated. The specimen of spinal fluid was slightly yellowish. It contained 2,250 mgm. per cent of protein and 45 mgm. per cent of sugar.

On May 22, 1943 three cc. of lipiodol were injected into the subarachnoid space and roentgenograms showed a complete block at L-2.

1st Operation. On June 4, 1943, a laminectomy from L-1 to L-3 inclusive was performed by the neurological surgeon in charge. When the dura was opened, a tumor was encountered. It measured $3 \times 1.5 \times 1.3$ cm. and was free except for attachment to a nerve root. After severing the nerve root proximal and distal to its fixation point in the tumor, the latter was lifted out *in toto.* Hemostasis was effected with Cushing silver clips and the dura was closed with a continuous cotton suture. The muscle layers were approximated with interrupted chromic catgut and the fascia with cotton sutures.

Histological Diagnosis. Perineurial fibroblastoma.

Course. Recovery was uneventful and the patient considered herself well until November 1944, about 17 months after the first operation. At this time she experienced a recurrence of the pain down the lower extremities associated with pain in the right hip which became quite severe.

Second Admission. She was readmitted to the Colorado General Hospital on June 1, 1945. Weakness of the lower extremities had progressed to the point where walking was considerably impaired. The Achilles reflex was still absent on the right, and there was marked hypalgesia in the L-3 distribution on both sides, being more marked on the left. There was some weakness of the bladder sphincter, resulting in dribbling.

Spinal puncture and Queckenstedt test did not reveal the presence of a block, but the needle was probably placed above the lesion. The specimen of fluid was not sent to the laboratory.

Lipiodol study was repeated and the roentgenogram showed a complete block at the level of L-4, the lower level of the block.

Impression. There was no question concerning the recurrence and progression of the

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* The spinal needle may have been placed just above the tumor. A later puncture at the 5th lumbar interspace revealed a block on the Queckenstedt test.

symptoms and signs indicating a compressive or destructive lesion at L-4, and the lipiodol study confirmed the fact. It was, however, difficult to conceive how a benign perineurial fibroblastoma which had been completely removed could recur.

2nd Operation. On June 26, 1945 the old wound was re-opened. At the level of L-2 and L-3 an extradural cyst was encountered, directly posterior to the dura. When it was incised, clear cerebrospinal fluid gushed forth. The inner aspect of the cyst presented a smooth glistening wall. The inside dimensions were about 2 × 3 cm. The outer aspect of the cyst was continuous with the surrounding scar tissue and muscle. The base of the cyst was attached to dura and severely compressed the latter ventralward. There was a tiny pin-hole fistula in the old line of incision in the dura. The fistula was directly adjacent to a silver clip which had been left in place on the cut edge of the dura at the first operation (Fig. 4). Spinal fluid could be

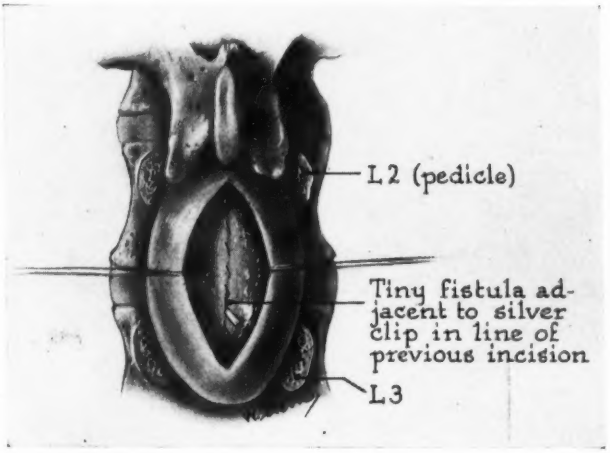

FIG. 4. Case 3. Drawing of the acquired extradural cyst, which has been incised so that one looks into it and at the dura forming its base. The cyst wall is formed of scar tissue which was continuous with muscle.

seen seeping through the tiny fistula into the cyst. The cyst wall was excised and freed from its attachment to the dura. The silver clip was removed and the dura resutured. Inspection of the cauda equina showed that there was no recurrence of tumor.

Course. Recovery was uneventful and at the time of discharge from the hospital on July 11, 1945 she had made remarkable improvement.

Pathology. The excised cyst wall presented the gross characteristics of scar tissue. The inner aspect was smooth and glistening, and the outer aspect was ragged. There were no separable layers or planes of cleavage.

Microscopically the inner wall was composed of dense fibrous (scar) tissue and appeared to be lined by mesothelium. The wall of scar tissue was continuous with surrounding muscle (Fig. 5), and should be compared with the well differentiated cyst wall in Fig. 2.

A small or "daughter" cyst was found in the wall of the large cyst. It is interesting because it reveals to some extent the mechanism of formation of such cysts. The cavity becomes enclosed by a wall of scar tissue, and the muscle contained within the cavity breaks apart. The fibers separate and fragment. The elements of inflammation and necrosis are not demonstrable (Fig. 6).

Case 4. #42-7656. O.C., a white male, was first admitted to the University Hospital, Iowa City, Iowa, on July 3, 1942. He had been progressively losing strength in the upper and lower extremities for 18 months. For 6 months he had been unable to walk. He had almost completely lost control of bowel and bladder function.

Examination revealed the following significant positive signs: Some stiffness in the neck. Loss of voluntary movement of the lower extremities with spasticity, hyperactive deep reflexes and Babinski sign on both sides. The general strength of the left hand was about 50 per cent of that on the right, and there was atrophy of the intrinsic muscles of both hands. All

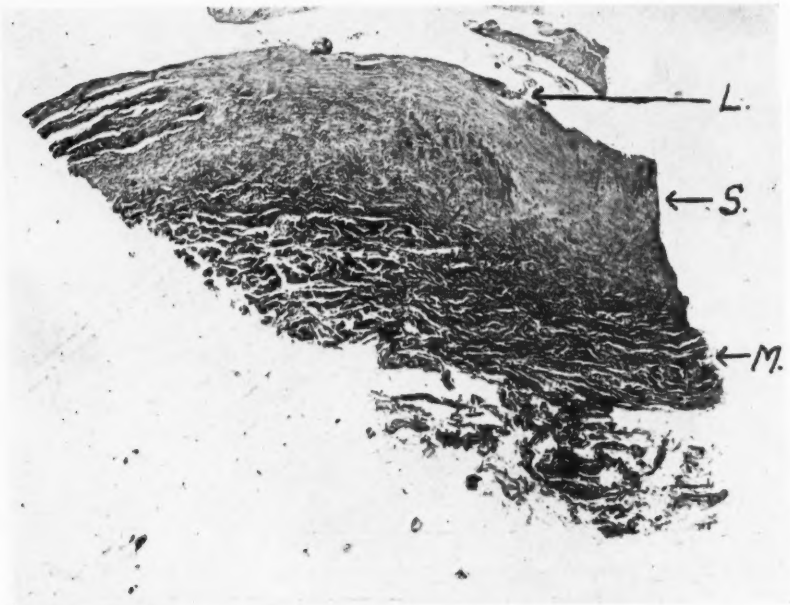

FIG. 5. Case 3. Photomicrograph of cross section of wall of acquired extradural cyst (×60). It is composed of a dense layer of scar tissue, S, which is continuous with muscle, M. The inner aspect of the cyst, L, appears to be lined with mesothelium, a strip of which has been torn loose at the arrow point. Compare with the well differentiated cyst wall in Fig. 2.

modalities of sensation were markedly impaired up to the level of the nipples, and there was patchy analgesia on the left hand and forearm.

Spinal puncture revealed an almost complete block on the Queckenstedt test and the fluid was xanthochromic. Lipiodol (5 cc.) was injected into the subarachnoid space by cisternal puncture, and a roentgenogram showed that there was a complete block at the level of the 5th cervical vertebra.

1st Operation. Upon the diagnosis of a cervical spinal tumor a laminectomy was performed on July 4, 1942. The laminal arches from C-4 to D-2 inclusive were removed. A large rather vascular meningioma was uncovered. It extended from the level of C-5 to D-1. It was extradural and to the left side of the cord, severely compressing the latter to the right. It was removed with some difficulty since it had extended some distance through the intervertebral canal at C-7 on the left. The 8th cervical and 1st thoracic roots on the left were imbedded in the tumor to such an extent that it was necessary to sacrifice them. As near as could be ascer-

tained by gross inspection, I felt I (O.H.) had removed all of the tumor, but due to its friable nature and the necessary piecemeal removal judgment could not be conclusive. A slight tear was made in the dura where the 7th cervical root on the left joins the spinal dura. The rent was in the axilla of the root, and although a suture was placed, the repair was not water-tight.

Histological Diagnosis. Angioblastomatous meningioma.

Course. The wound became infected with *Staphylococcus aureus*, and spinal fluid drained from it for almost 3 weeks. It then healed without further complication, and he was discharged on Aug. 3, 1942. He made rapid and marked recovery from the paralysis to the point where he

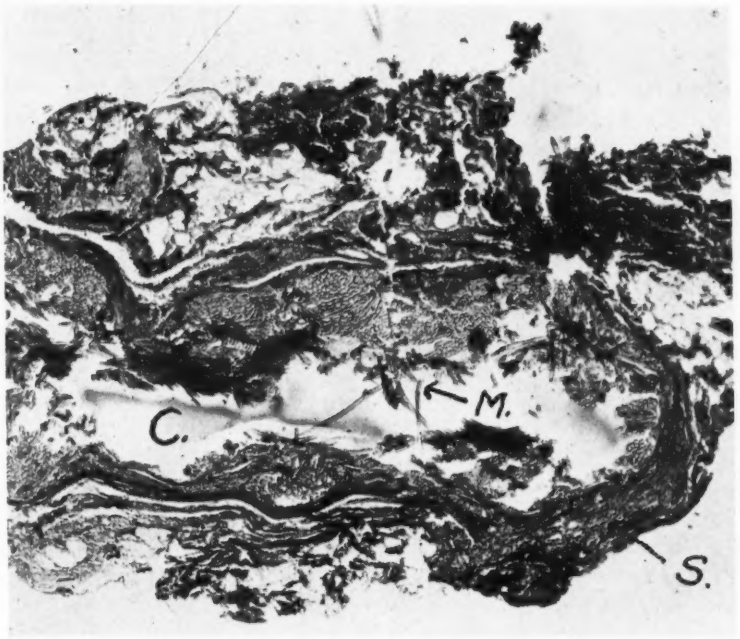

FIG. 6. Photomicrograph of a tiny cyst in formation located in the wall of the cyst described in Case 3 (×45). S, scar tissue wall, C, cyst cavity in formation. Note muscle fibers, M, undergoing separation and fragmentation.

could walk without aid. In January 1943, about 6 months after removal of the tumor, the former symptoms and signs of paralysis began to recur.

Readmission. On Mar. 24, 1943 he was readmitted to the hospital in much the same condition as he presented on the 1st admission.

2nd Operation. Under the impression that the tumor had recurred, the wound was re-explored. A cyst posterior to the dura was disclosed. It was about the size of a golf ball, and when it was incised, clear spinal fluid gushed forth indicating that it was under considerable tension. The inner lining of the cyst was smooth and glistening, and its outer aspect was continuous with scar tissue and muscle. A small pin-hole fistula was found at the axilla of the 7th cervical root on the left where the dura had been torn at the first operation. The fistula admitted spinal fluid to the cyst but did not allow its return to the subarachnoid space. The fistulous opening into the cyst was somewhat slit-like with compresssible lips strongly reminiscent of

the ureteral orifice in the urinary bladder. The cyst wall with adjacent scar tissue was dissected out. The fistula in the dura was excised and the latter repaired. It was obvious that the cyst had severely compressed the spinal cord and was responsible for the recurrent paralysis. A careful search revealed no evidence of recurrent tumor.

Microscopically, as in Case 3, the cyst wall was found to be composed of dense fibrous tissue with probably a mesothelial lining. There was no evidence of inflammation.

Course. The patient again made considerable improvement for 4 months at which time the symptoms and signs of paralysis of the upper and lower extremities began to recur for the second time. The patient is no longer under the care of one of the authors (O.H.), but re-exploration was advised. It has not yet been done. Since there was no evidence of recurrent tumor at the second exploration, it is possible that another extradural cyst has developed by the same mechanism that was responsible for the first.

Discussion. It is clear that the causal factor for the recurrence of symptoms and signs in these two cases was an acquired extradural cyst. The cyst was a natural consequence of a persistent tiny fistula leading from the spinal subarachnoid space. It was also clear on close inspection that the silver clip on the free edge of the dura in Case 3 was responsible for the persisting fistula inasmuch as the dural margins could not seal completely about the foreign body.

It is easy to conceive how spinal fluid could find ingress into the cyst cavity and be forced into it when the patient strained, coughed, sneezed or did anything that would raise the spinal pressure. It was equally obvious that the intracystic fluid would have considerable difficulty in finding egress back into the subarachnoid space through the fistula. In short, such a fistula serves as a one-way valve with the consequence that fluid accumulates in the cyst cavity under ever increasing pressure.

It is a well known fact that with a persisting spinal fistula admitting spinal fluid into the soft tissue, the accumulation of fluid soon becomes encased by a non-absorbing wall. The smooth glistening appearance of the wall suggests a mesothelial lining. It is because of this development that the construction of fistulas between the ventricles or subarachnoid spaces and soft tissues are not practical or of permanent value in the treatment of congenital hydrocephalus.

It is probably the rule that closures made in the dura are not immediately water-tight, and if a drain is left in the soft tissue wound for a day or two, considerable spinal fluid will escape from the wound. The flow will usually cease in 3 to 5 days, indicating that the dural wound has healed sufficiently to become water-tight. If the wound is not drained, spinal fluid no doubt escapes into the soft tissues. The latter, and particularly muscle, will absorb a considerable amount of the fluid over a period of days. Eventually, however, if fluid continues to escape into the soft tissues, it will become encysted by a non-absorbing wall. In the 2 cases presented here the circumstances were such that fluid accumulated in the cyst under increasing pressure. Hence the cyst assumed considerable significance because by compressing the cauda equina or the spinal cord at the site from which a neoplasm was removed, it was responsible for a recurrence of the previous symptoms and

signs. Sufficient time had elapsed after the operation to arouse a suspicion that the tumor had recurred. Since the tumor in both of these cases was benign, there was no hesitancy in advising re-exploration. It is conceivable, however, that similar cases might be encountered where the pathology is that of malignancy or is doubtful. In such a case, the recurrence of symptoms 4 or 5 months after operation might provoke the impression of a hopeless prognosis and advice against re-exploration. In any case, since the development of an acquired extradural cyst, such as reported here, is a distinct possibility, it should be given proper consideration in diagnosis. Certainly if intradural operations are done for reasons other than removal of a neoplasm and the symptoms and signs of cord compression later develop, this lesion should be included among the possible causes.

Fortunately, it is very uncommon for a subarachnoid fistula, either intracranial or intraspinal, to persist in the absence of infection and dural wounds normally heal. The results of the experience reported here teach that dural tears or incisions about the junctional zone of a root, and especially in its axilla, may not heal so readily. It also becomes rather obvious that leaving silver clips on the sutured margin of the dura, especially the spinal dura, is bad practice and moreover unnecessary.

SUMMARY AND CONCLUSIONS

One case of spinal extradural cyst, congenital, is reported. It is the 22nd case on record. In essentially all of its clinical characteristics it conformed with the other cases reported. The early onset of symptoms and signs of an intraspinal compressive lesion at about the mid-dorsal region associated with the roentgenological findings of kyphosis or scoliosis and widening of the spinal canal is sufficient to warrant a consideration of the lesion. A study of the pathology indicates that the cyst probably arises from a misplaced rest of cells. If the onset of paralysis is insidious and the cyst is removed, the prognosis should be good.

A case of spongioblastoma of the spinal cord is presented in relation to differential diagnosis because the case was indistinguishable clinically from the syndrome of spinal extradural cyst.

Two cases of acquired extradural cyst are reported. One developed in the low cervical region, manifesting itself about 6 months after removal of an extradural meningioma. The cause was a small persistent fistula in the axilla of the junction of a nerve root and spinal dura.

In the other case a cyst developed in the lumbar region, manifesting itself about 17 months after removal of an intradural perineurial fibroblastoma. The cause was a small persistent fistula in the old line of dural incision and at a point where a silver clip had been applied to the cut margin of dura and allowed to remain when the latter was sutured.

Cases of this kind, though probably rare, are instructive. If symptoms and signs of an intraspinal compressive lesion occur or recur after an operation in which the subarachnoid space is opened, one should consider the

possibility of acquired extradural cyst. The lesion is benign and the prognosis good after its removal and repair of the fistula leading into the subarachnoid space.

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THE LUCITE CALVARIUM—A METHOD FOR DIRECT OBSERVATION OF THE BRAIN

II. CRANIAL TRAUMA AND BRAIN MOVEMENT*

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INTRODUCTION

IN A PREVIOUS report[23] an experimental surgical technic in which the convex portion of the monkey skull is replaced by a transparent lucite calvarium was described. Because a large area of both cerebral hemispheres is exposed through the lucite window, the method is ideally suited for the study of intracranial phenomena under "closed box" conditions. It may be of interest to mention that the technic was developed primarily to study the effects of cranial trauma on the underlying brain. The patterns of brain motion resulting from blows to both the freely movable and the immobile head were recorded by high-speed cinematography.

It is stressed that these experiments were conducted to study the motion patterns of the cerebral mass and any other visible physical change. It is not a study of cerebral concussion. Although brain movement and concussion are probably closely interrelated the mechanism of concussion is not explainable in the light of our findings. It is hoped, however, that this study may furnish additional data toward the eventual solution of the problem of cerebral concussion.

HISTORICAL BACKGROUND

A review of the literature of the past century and a half discloses that the efforts of investigators have been directed principally toward explaining the phenomenon of concussion. Comparatively few studies have been undertaken with a primary objective of explaining the mechanics involved in the production of focal and contrecoup brain injury. In establishing the historical background for this presentation these studies of cerebral movement and damage are emphasized. Reference to studies of concussion is made only insofar as these studies contribute to the subject under discussion. Excellent reviews of the development of our current knowledge of cerebral concussion may be found in the writings of Polis,[20] Miller,[18] Denny-Brown and Russell,[7] and Denny-Brown.[6]

According to Polis,[20] Fallopius was first to describe cerebral damage on the side of the brain opposite the site of impact. However, the term "contre-

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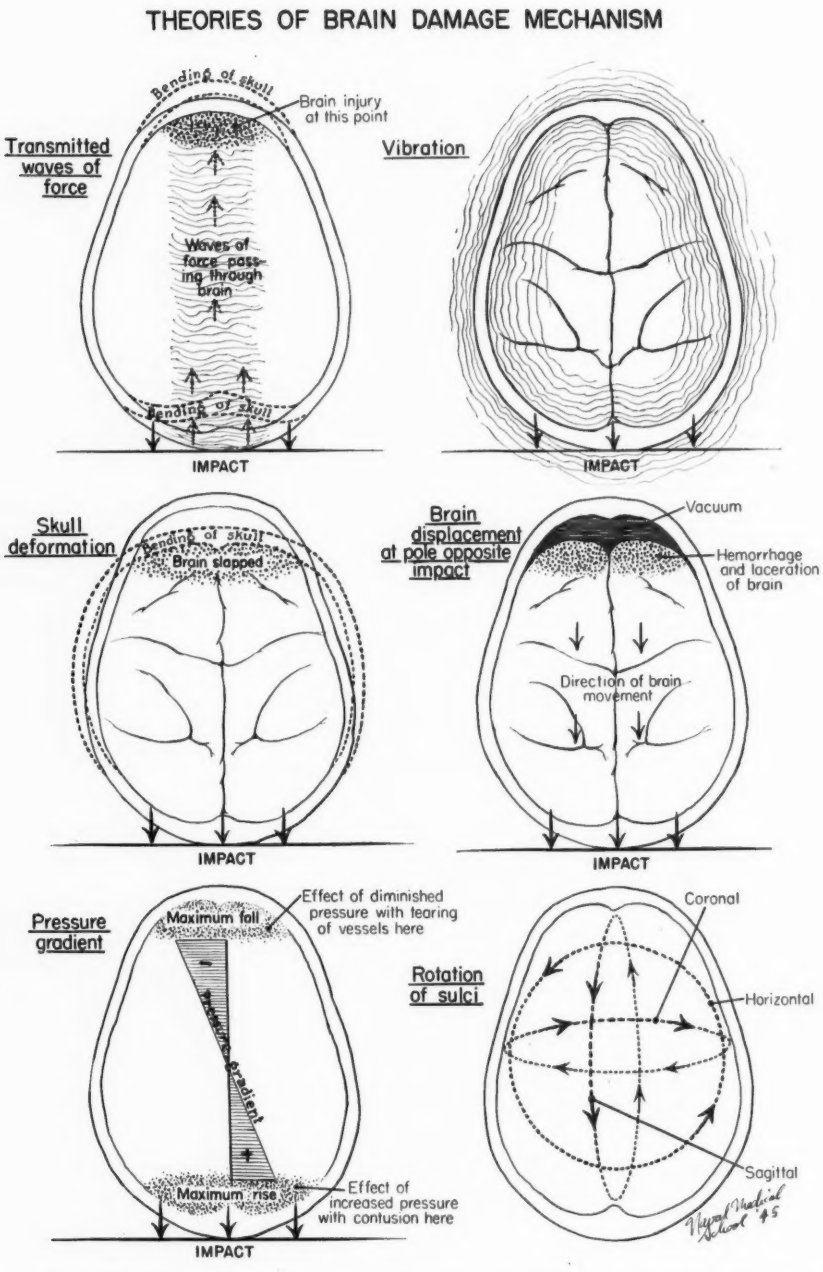

FIG. 1. Theories concerning the mechanism of coup and contrecoup cerebral damage. The skull deformation shown in the diagram representing the theory of transmitted waves of force has not been described in all of the writings on this theory but is included for the sake of simplicity.

coup" did not come into popular use until 1766, at which time a series of papers on the subject was presented by various members of the Paris Academy of Surgery. Since this time many explanations of the mechanism of focal and contrecoup cerebral damage have appeared in the literature. These theories, which may be classified under six separate mechanisms (Fig. 1), explain brain damage on the basis of: (a) vibrations transmitted from the skull to the underlying brain; (b) passage of waves of force through the brain; (c) brain displacement at the pole opposite the site of impact; (d) deformation of the skull; (e) pressure gradients set up within the brain as a result of the blow; (f) rotation of the cerebral mass within the confines of the skull.

Vibration Theory. This theory, which is the oldest, was originally formulated in 1766 as the consensus of the various members of the Paris Academy of Surgery who postulated the mechanism of contrecoup damage. According to these observers the brain damage was attributed to vibrations originating in the skull at the site of the blow and transmitted throughout the entire skull, arriving at a point diametrically opposite the site of origin. Depending on their intensity, these vibrations would or would not fracture the skull at the contrecoup point but the brain at this point would supposedly be subjected to a force greater than in other areas.

In 1835 Gama[12] gave a new impetus to the vibration theory by conducting the first experiments designed to explain contrecoup lesions. His investigations were carried out on models consisting of glass flasks containing ichthyocol in which colored threads were dispersed. When the flask was struck the threads vibrated, the pattern of vibration depending on the site and force of the blow. The vibration theory was short-lived but as late as 1888, Bryant[2] noted that a severe blow on the head may not fracture the skull but nevertheless may cause vibrations that are transmitted to the brain and lacerate its substance.

Theories Based on Transmitted Waves of Force. In the latter part of the nineteenth century the first theories explaining contrecoup on the basis of transmission of waves of force through the brain substance made their appearance. In 1873 Félizet[10] demonstrated the momentary depression of the skull resulting from blows by dropping skulls filled with paraffin and coated with ink onto a hard surface. The resulting mark on the surface struck was larger than any possible point of contact of the calvarium and could only be explained by flattening of the skull.

These findings were elaborated in 1878 by Duret[9] who explained contrecoup as due to a bulging outward of the skull at the pole opposite the point depressed by the trauma. The bulging outward (soulevement) produced a vacuum which ruptured surface blood vessels. The studies of Miles[17] published in 1892 were purported to confirm Duret's theory. This observer coated one side of the heads of fresh cadavers with vaseline or putty and struck the opposite side. The resulting indentation of the putty and vaseline suggested a "cone of bulging." He therefore concluded that the vacuum

produced beneath the bulging tore the superficial cerebral vessels. Miles further noted that Erichsen believed contrecoup damage was due to a wave of force being transmitted through the brain substance and "breaking" against the inner table of the skull on the opposite side.

In 1895 Chipault and Braquehaye[4] employed a tambour arrangement to demonstrate momentary rises and falls in intracranial pressure at the sites and opposite points of blows respectively. The tambours were screwed into the skulls of large dogs and the detached heads of fresh cadavers. These findings have been confirmed recently by Walker, Kollros and Case[25] with an improved method of manometric recording.

Courville's recent report[5] lends additional support to the theory of transmission of lines and waves of force through the brain. According to this observer the head in motion resembles a bullet in flight since both have a line of force in their trajectory. When the forward motion of the head is suddenly arrested the line of force is immediately reversed and extends backward through the nervous tissue to a point opposite the site of impact. Presumably, waves of force spread outward from the main line of force. The lines and waves of force are modified by the angle of impact and by the deflection and diffusion resulting from their contact with structures of different densities, such as the falx and tentorium. Courville concludes that the "ultimate lesion depends on the relation of the waves of force along this line to the cortex and the subcortex which lie against the inflexible bony wall, on the degree of resilience of the blood vessels in the areas affected by the waves of force and on the anatomic structure of the brain in the area involved." In support of his theory Courville mentions that cerebral lesions are occasionally demonstrable along the path of force at autopsy.

Brain Displacement at Pole Opposite Impact. Russell, in 1932,[22] objected to the theory of transmitted force. He recognized that trauma caused movement of the brain within the skull but held that contrecoup was due to the brain tearing itself away from its covering by the force of its own momentum. Thus, when the moving head is suddenly arrested the brain at the pole opposite the point of impact is moving in a forward direction whereas the skull has been arrested. The degree of skull and brain damage is proportional to the suddenness with which the momentum is altered.

In their classical experiments on cerebral concussion published in 1941, Denny-Brown and Russell[7] note that contrecoup lesions occurred infrequently but attributed them to the fling of the brain creating a momentary vacuum under the membranes opposite the point struck.

Similarly, Rowbotham[21] and Dott[8] describe an area of diminished pressure at the pole opposite the site of impact due to movement of brain away from skull. The suction created by this movement may rupture not only the surface vessels but also those more deeply placed.

Contrecoup Injury by Skull Deformation. This theory, which is related to the vibration theory, explains contrecoup damage on the basis of flattening of the skull at the time of impact. Skull deformation, as noted above, had

been demonstrated experimentally by both Félizet and Miles. According to Munro[19] the mechanism is as follows:

> With the skull and brain both in motion in the same direction, the brain lags slightly behind the skull, because of a difference in inertia. When the skull is struck and its motion arrested, the force of the blow is transmitted throughout the bone. The flattest arch in the skull leading from the point of impact is mechanically the weakest. Its apex will either bend outward or break and allow the distal extremity to move inward in the direction of the center of the head. The lag in the forward progress of the brain within the skull places it momentarily where the inward shift of the bone collides with and, as it were, slaps its surface. The amount of damage done will depend upon the inertia of the brain, the weakness of the bony arch and the abruptness with which motion is arrested.

This explanation is likewise offered by Rowbotham in addition to the vacuum theory.

Pressure Gradient Theory. This theory of contrecoup was proposed by Goggio[13] in 1941. He draws objections to both the compression wave and elastic body theories. His objection to the former theory is that the brain damage should always be greatest at the site of origin of the wave of force. He furthermore objects to alternate lengthening and shortening of the skull as the explanation of cerebral damage because the bony buttresses do not make it an elastic body and because contrecoup occurs in the presence of fractures which should lessen the deformation. The pressure gradient theory treats contrecoup as a problem in intracranial hydrodynamics in which the maximum fall in intracranial pressure occurs at a point diametrically opposite the point of impact. The sudden diminution of pressure in the contrecoup area causes rupture of blood vessels.

Rotation Theory. Although rotation of the cerebral mass as a whole was suspected as early as 1865 its relationship to contrecoup was not clearly explained and fully appreciated until Holbourn[15] presented his physical interpretations in 1943.

In 1865 Alquié[1] conducted experiments designed to test the vibration theory which had been supported by the experimental work of Gama in 1835.[12] Alquié conducted his studies with glass models filled with gelatin, with dry skulls, and with the skulls of cadavers and live animals. He observed that a blow caused a centrifugal rotation of the brain which flung it against the inner table of the skull, particularly in the direction of the blow, following which there was a rebound. The surface convolutions sustained the greatest damage since they were subjected to the greatest rotary force.

Further evidence for cerebral mass movements was reported by Ferrari[11] in 1882. This observer filled skulls with gelatin and cerebral tissue in which glass capillary tubing or cover glasses were embedded at various depths. When these models were struck on the vertex with falling weights, the glass objects near the surface of the brain were fragmented even though the skull was not fractured.

It is noteworthy that many writers on the subject of contrecoup have recognized that the brain moved with relation to the skull. Goggio, in particular, stressed the rotation of the brain within the skull as an explanation for

the increased frequency of the brain damage in the frontotemporal region. Others have recognized this movement and used it to explain the tearing of the cortical veins from the longitudinal sinus leading to the development of a subdural hematoma.

Holbourn's explanation of contrecoup is based on the physical principles applicable to confined masses of cerebral density and elaborated by experiments on gelatin models. He believes that brain damage is due to rotational acceleration forces and not to transmitted waves of compression and rarefaction. He states that the prime requisite for contrecoup damage is that the head be set in rotation by the blow. This rotation may be in the coronal, sagittal and horizontal planes or their combinations. The rotation is transmitted to the brain which glides in its dural compartments. The gliding motion is relatively free except where the brain is confined by bony structures, namely, in the frontal and middle fossae of the skull. In these areas shear strains occur which lacerate cerebral tissue and tear blood vessels. Damage is less in those areas of the brain that are free to glide, or under conditions where rotation is minimal. Holbourn's theory accounts more satisfactorily than any of the others for the distribution of contrecoup lesions as seen in fatal cases of head injury. It is also supported by the direct observations of brain movement as seen in the high speed motion pictures of this study.

Gunshot Wounds of the Brain. Although many writings on experimental gunshot wounds of the brain appear in the literature, the background for our study was established by the studies of Butler, Puckett, Harvey and McMillen.[3] By means of both high speed cinematography and a microsecond roentgenographic technic these observers had demonstrated that the passage of a high velocity missile through the heads of experimental animals produces a large, rapidly expanding and pulsating temporary cavity within the brain. The pressure within this cavity increases in direct proportion to the velocity of the missile and may be sufficient to cause extensive comminution and disarticulation of the bones of the skull. These workers furthermore demonstrated that the extensive fracturing of the skull was due to the high radial velocity imparted to the cerebral tissue. When the brain was removed through the foramen magnum prior to the wounding only minor skull damage occurred.

METHODS AND MATERIALS

Macaque monkeys fitted with lucite calvaria were used in all of the experiments. The method for preparing the experimental animals was described in the previous report.[23] Since this, several improvements in technic have been introduced.

Most important of the technical changes is that the holes originally placed to drain the space between the lucite plate and the brain are no longer used. The xanthochromic fluid that forms in this space is absorbed within a 7–14 day period. An additional important step is the re-suturing of the scalp with tantalum wire (0.007 inch) after each observation of the brain rather than resecting the scalp at the end of the second stage. By this means the incidence of wound and meningeal infection has been appreciably lowered and the method better adapted to a long period of observation. Finally, the lucite calvarium is now fastened to the

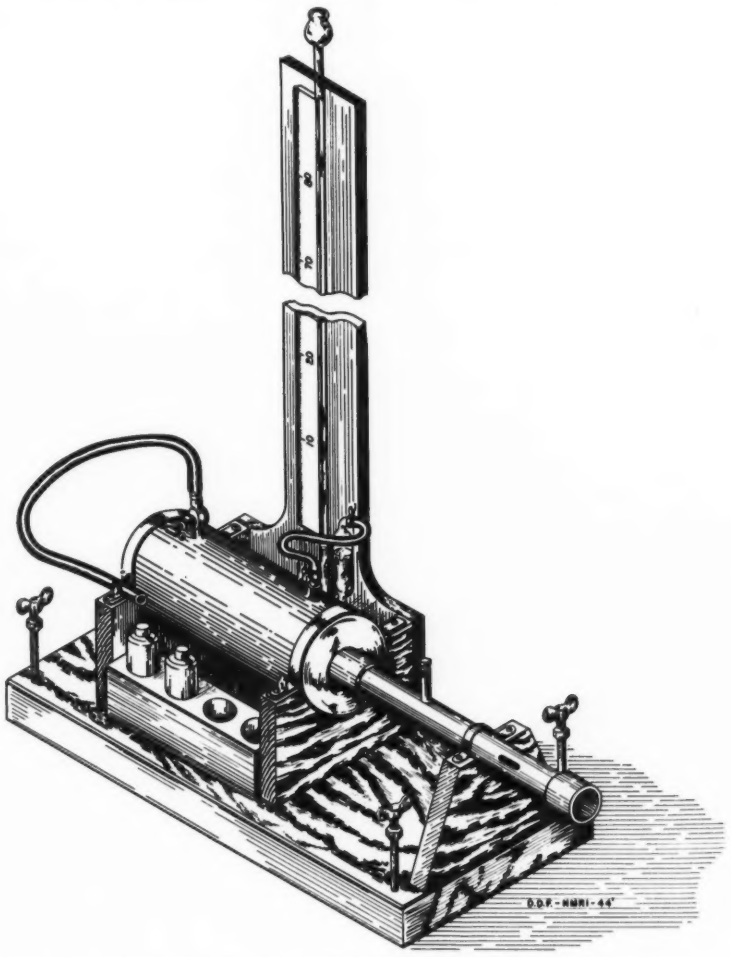

FIG. 2. Compressed air gun designed for delivering calibrated blows to the head. Note the vertical mercury manometer, the compressed air chamber, the horizontal barrel through which the projectiles are propelled and two of the four projectiles in the front rack. The vents near the end of the barrel release the compressed air charge behind the projectile and thereby permit a sharp, instantaneous blow to be struck.

skull with the standard 5/16 inch vitallium skull screws rather than the $\frac{3}{8}$ inch bone screws which have a much larger diameter.

Method for Delivering Blows to the Head. Blows on the head were delivered by means of a special apparatus operating with compressed air which was designed for this study by Captain R. H. Draeger (MC), U.S.N. The apparatus (Fig. 2) has four essential parts:

1. A cylindrical chamber that contains the compressed air charge.
2. A vertical mercury manometer for recording the air pressure.

3. A horizontal barrel leading from the chamber through which projectiles are propelled.
4. Cylindrical aluminum projectiles weighing 100, 200, 300, and 500 gm.

Compressed air can be obtained from either the usual laboratory outlet or from a cylinder. The charge is built up gradually behind the projectile which is suddenly released by a trigger mechanism. Air vents in the end of the barrel release the driving pressure and allow an instantaneous blow to be struck.

The velocity, energy and momentum of each projectile at a given air pressure were calibrated with a ballistics pendulum at the Naval Research Laboratory, Anacostia, D. C. The force of the blow can be considerably altered by varying the weight of the projectiles and pressure. The maximum blows delivered by the apparatus have been calibrated at a pressure of 800 mm. mercury (Table 1).

These maximal values for the 100, 200, and 300 gm. projectiles exceed the velocity of 28.3 ft./sec. and energy of 17.83 ft./lb., which Denny-Brown and Russell found necessary to produce concussion in the cat and monkey.

Due to its lightness in weight and portability the apparatus is ideal for experiments that require working in different laboratories. A further advantage is that by varying the weights

TABLE I

Velocity and energy of blows at a pressure of 800 mm. mercury

Weight of Projectile (grams)	Velocity (ft./sec.)	Energy (ft./lb.)
100	77	20.4
200	57	22.4
300	44	20.0
500	34.5	16.3

of the projectile and the compressed air pressures one is able to deliver blows of constant velocity and variable energy or vice versa.

In all of the experiments recorded in this investigation a blow having a velocity of 30 ft./sec. and an energy of 6.6 ft./lb. was used. This blow was obtained by propelling the 200 gm. projectile with a charge of air compressed to 300 mm. mercury. In repeated experiments on conscious animals this blow failed to cause any objective evidence of concussion. It is therefore a subconcussive blow. In the cinematographic studies the blows were delivered to the frontal, temporal, parietal, and occipital regions of the rigidly fixed or freely movable head. Fixation of the head was obtained by binding it to a heavy steel angle-iron with adhesive tape. The angle-iron, in turn, was bolted to the rigid oak frame of the animal holder. By this means the head was immobilized satisfactorily although not perfectly.

Cinematographic Recording of the Blow. Cinematographic recordings of the pattern of brain movement resulting from the blow were made on 16 mm. color film with an Eastman High Speed Camera. In these experiments a rate varying from 2000 to 3000 frames per second was used. Since intense illumination is necessary to obtain proper film exposure, a battery of two 13,800 watt and two 16,000 watt arc lamps was used to illuminate the animal's head. The intensity of the illumination could not be measured but was estimated at 3,000,000–4,000,000 foot candles. The blow was timed to fall within the period of maximum camera speed.

The pattern of brain movement was analyzed in a Movieola and by repeated projection of a film loop on the screen. Animated drawings were made from the films of one blow so that the movement pattern could be analyzed in greater detail. When these films are projected at a speed of 16 frames per second the brain movement is prolonged from 125 to 185 times its normal duration. Even under these conditions of slow motion the movement does not occupy more than a few seconds and it is difficult to be certain of the finer patterns of motion.

Gunshot Wounds of the Brain. Observations of the effects of perforating missiles on the

brains of two monkeys were made in the laboratories of the Department of Biology of Princeton University. The perforating missiles consisted of steel spheres, $\frac{1}{8}$ inch in diameter, weighing 130 mgm., which were fired at velocities of 1121 and approximately 1800 ft./sec. Perforating bitemporal wounds were obtained in both instances. The path of the missiles was identical in both instances (Fig. 3).

The cinematographic recording and analysis of the trauma was carried out according to the method described for the blow experiments excepting that black and white motion picture film was used.

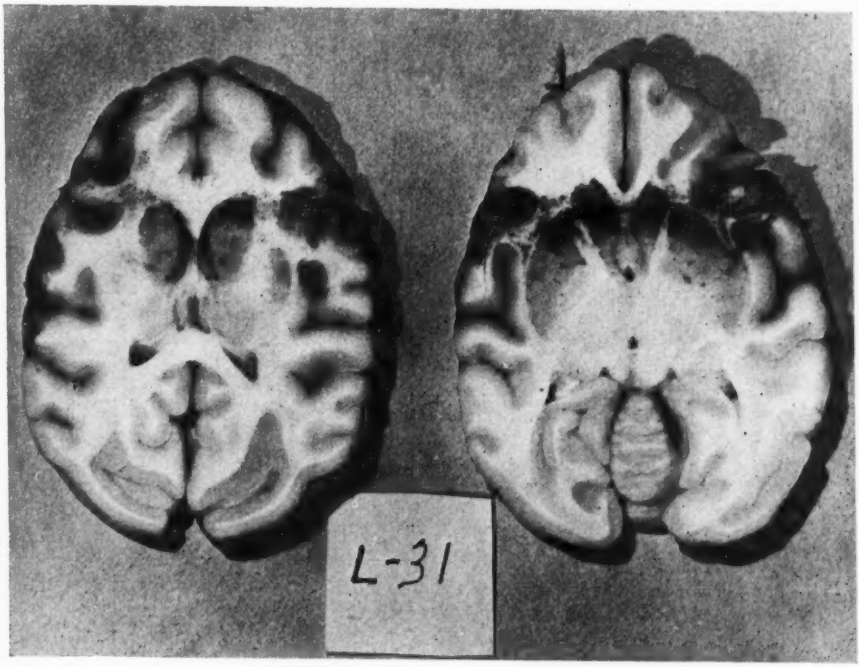

FIG. 3. Perforating gunshot wound of the brain with an $\frac{1}{8}$ inch steel sphere having a velocity of approximately 1800 ft. per sec. Note the hemorrhagic softening along the path of the missile.

RESULTS

It was clearly demonstrated that the subconcussive blows used in this study gave rise to gliding movements of the brain within the cranial cavity. The brain movement lagged behind the motion of the skull, apparently due to the inertia of the cerebral mass.

Movement of the brain and skull is probably most clearly visualized if both are considered as spheroid. As such both are subject to motion in a horizontal, coronal or sagittal plane or combinations thereof (Fig. 1). The skull rotates on the cervical axis whereas rotation of the cerebral mass occurs within the cranial cavity.

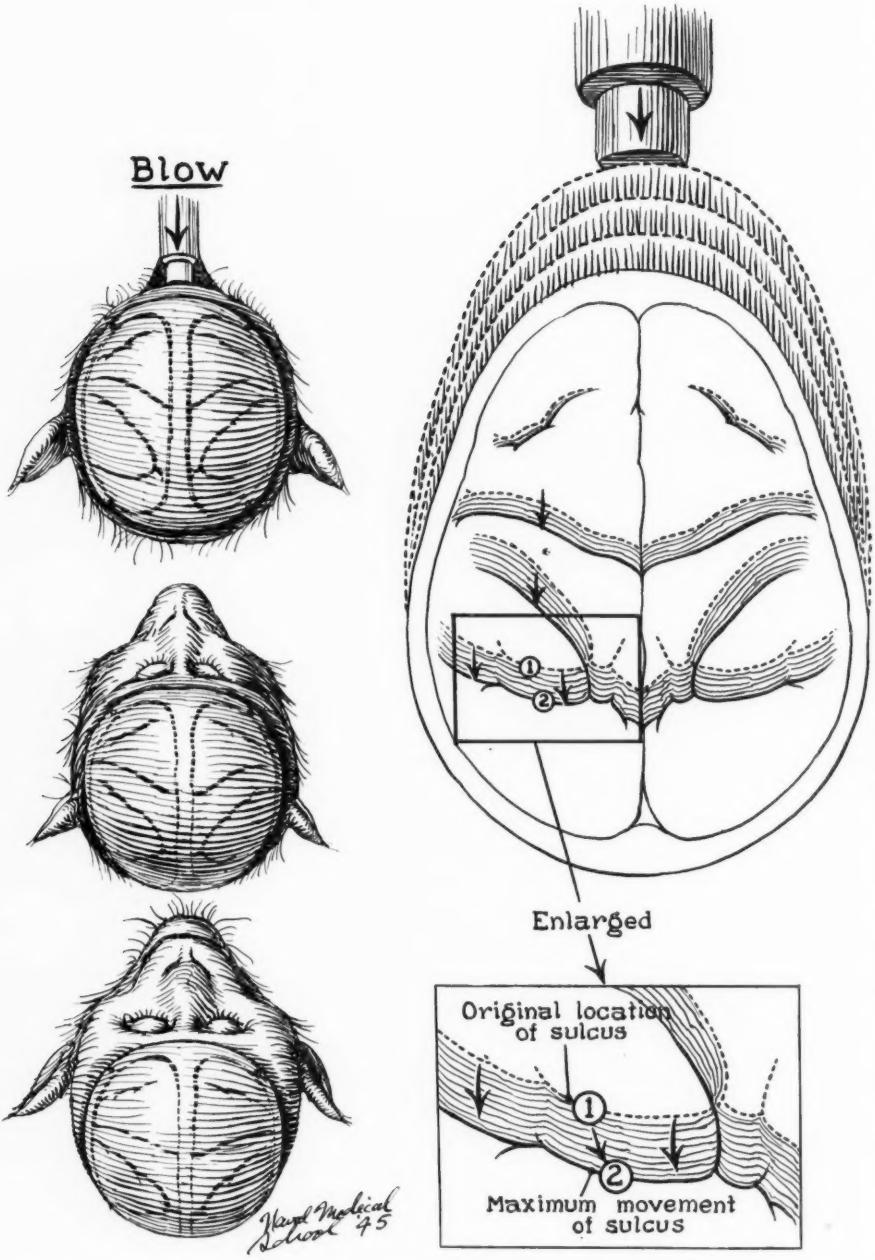

FIG. 4. Head displacement and convolutional rotation patterns resulting from midline frontal blows.

In this study the rotatory movements of the cerebral convolutions were chiefly in a sagittal or horizontal plane. Distinct movements through a coronal arc were not seen. This is apparently due to the falx cerebri serving as a baffle between the lateral compartments of the cranial cavity.

The degree of convolutional glide varied considerably in the different areas of the brain. It was invariably of greatest amplitude in the parietal and occipital lobes, irrespective of the site of the blow. Frontal lobe motion, while present, was minimal. Movement of the temporal lobe could not be seen through the lucite plate.

Movement of the brain was affected considerably by the mobility or immobility of the head. A blow that routinely caused a substantial amount of convolutional displacement in the freely movable head produced little or no movement when the head was immobile. This point was demonstrated repeatedly.

An additional observation of interest was the dampening effect of cerebrospinal fluid pressure on brain displacement. An identical blow delivered to the head of the same animal after withdrawal of cerebrospinal fluid was attended by considerable increase in the amplitude of convolutional glide.

Perforating gunshot wounds of the brain caused a sudden outward displacement of the cerebral mass without visible rotation evidenced by flattening of the convolutions against the inner surface of the lucite plate.

Each of these findings is elaborated in the ensuing data.

Midline Frontal Blows. The pattern of brain movement resulting from blows in this area was visualized from two directions. In two experiments the camera was focused on the convex surface of both hemispheres. In the third trial the head was photographed in profile so that only the cerebral hemisphere of the same side was visualized.

In each instance the midline frontal blow caused the head to be moved through a sagittal plane of rotation with the neck serving as a pivot (Fig. 4). The rotational glide of the surface convolutions was through the same plane.

Because of the inertia of the brain described above there was a momentary pause before the surface convolutions followed the skull through the sagittal plane of displacement. However, before the head proper had begun its reverse motion to the starting position the brain had already commenced its anterior rotation. The final movements of the brain consisted of anteroposterior undulations of diminishing amplitude.

Maximal convolutional displacement occurred in the parietal lobes. Motion of the frontal convolutions, while present, was considerably less in degree.

When a blow was delivered to the frontal region of the immobilized head, brain motion was considerably reduced.

Midline Occipital Blows. Blows in this region of the freely movable head caused a rotational pattern which was the reverse of that noted with the midline frontal blows. Again convolutional glide was greater in the parietal region and minimal in the frontal lobes.

Parietal Blows. Since blows in the parietal region cause a displacement of the head in a combined horizontal, coronal and sagittal plane, movement of the brain is of a similarly complicated pattern. Brain movement was much greater following blows in the parietal and temporal regions than in the frontal and occipital regions.

Despite the displacement of the head through a coronal arc, a coronal rotation of the brain was not seen. In other words the cerebral hemisphere

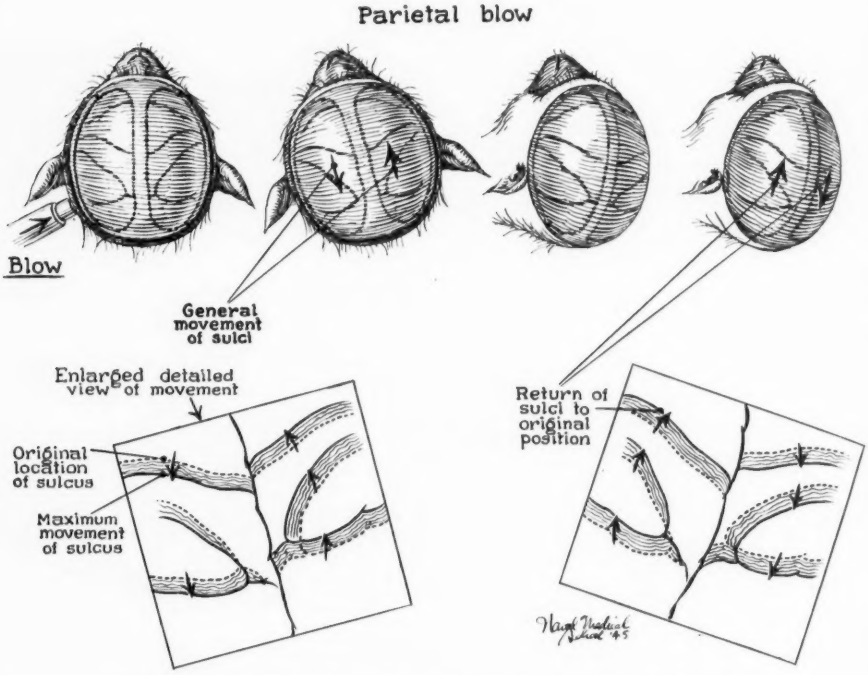

FIG. 5. Patterns of head and convolutional motion resulting from parietal blows.

on the side of the blow was not flung against the falx nor was the contralateral hemisphere displaced away from it. Cerebral rotation occurred principally in a horizontal plane. When the head was displaced anteriorly and to the right side (clockwise) by blows in the left parietal region, the first movement of the brain was similarly clockwise. The reverse was true for right parietal blows. Following the original displacement the rotation of the brain was counterclockwise, finally ceasing after diminishing clockwise-counterclockwise oscillations (Fig. 5). Convolutional motion was invariably greater in the parieto-occipital region than in the frontal region but was equally extensive in the two hemispheres.

The decreasing oscillations of the brain as it comes to a state of rest are particularly striking with the parietal and temporal blows. These settling movements are especially prominent in the parietal lobes, where maximum cerebral movement is located. During the phase of greatest brain rotation there is considerable stretching of the surface veins. In two ex-

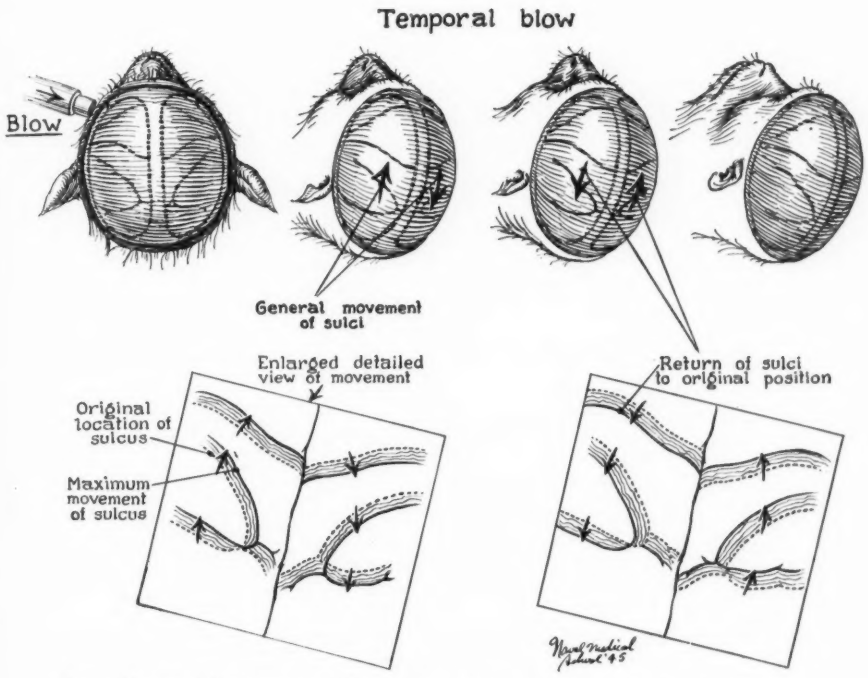

FIG. 6. Nature of displacement of the head and of the cerebral convolutions resulting from temporal blows.

periments parietal veins entering the longitudinal sinus were torn, leading to the development of a subdural ("sublucite") hematoma.

Following a parietal blow in one animal the intracranial pressure was reduced by removing 2 cc. of cerebrospinal fluid by lumbar puncture. This is equivalent to the removal of approximately 20 cc. in humans. Repetition of the blow under these conditions caused a considerable increase in the amplitude of brain rotation. Furthermore, the final oscillations of the brain were especially striking in this case. Thus the role of the cerebrospinal fluid as a baffle was clearly demonstrated.

In contrast to the experiment in which a blow was delivered under conditions of reduced intracranial pressure were those in which blows were delivered to the immobile head. Under these conditions the amplitude of

brain movement was either considerably reduced or absent. The pattern of rotation, however, was similar to that which occurred with the freely movable head.

Temporal Blows. The pattern of brain movement with blows in this region was similar to that noted with the parietal blows. Because the amplitude of head rotation was greatest in the horizontal plane the brain rotation was likewise most marked in this plane (Fig. 6). Clockwise and counterclockwise horizontal rotation of the brain was initially dependent on the original displacement of the head. Blows to the temporal area of the freely movable head were followed by considerable cerebral motion. The final undulations of the brain were particularly prominent. In contrast, temporal blows to the fixed head caused only a slight rotatory motion, although the pattern of motion was similar.

Gunshot Wounds of the Head. In the two experiments in which the $\frac{1}{8}$ inch steel spheres traversed the frontotemporal region of the skull at velocities of approximately 1100 and 1800 ft/sec., the effects on the brain were similar but more intensified in the instance of the greater velocity. The considerable increase in intracranial pressure resulting from the passage of the missile caused a sudden flattening of the brain against the inner surfaces of the lucite plate and the skull. This flattening was particularly striking along the edge of the bone supporting the falx where a ridge of cerebral tissue formed momentarily on each side (Fig. 7). Considerable hemorrhagic softening was noted along the path of the missile when the brain was cut in horizontal section.

The gunshot wounds caused no visible rotation of the cerebral hemispheres. If rotation did occur, it was masked by the sudden outward displacement of the brain. The lucite plate remained firmly fixed to the skull following both woundings although in the second experiment it was extensively fractured at the point of exit.

DISCUSSION

In an experiment of this type it is necessary to determine if the data accumulated from observations on the experimental animals may be interpreted as representing the conditions that exist in the human being. We believe that this is permissible because:

1. The intracranial anatomic relationships are similar in the macaque and the human skull.
2. The "closed box" conditions of the cranial chamber have been maintained.
3. The cushioning of the brain by the cerebrospinal fluid was preserved.
4. Removal of the dura overlying the convexity would not permit motion not normally present since the brain is not attached to it except when the cortical veins enter the sagittal sinus. The smooth inner surface of the dura provides an ideal surface on which the convolutions can glide.

Movement of the brain resulting from blows on the head has been clearly

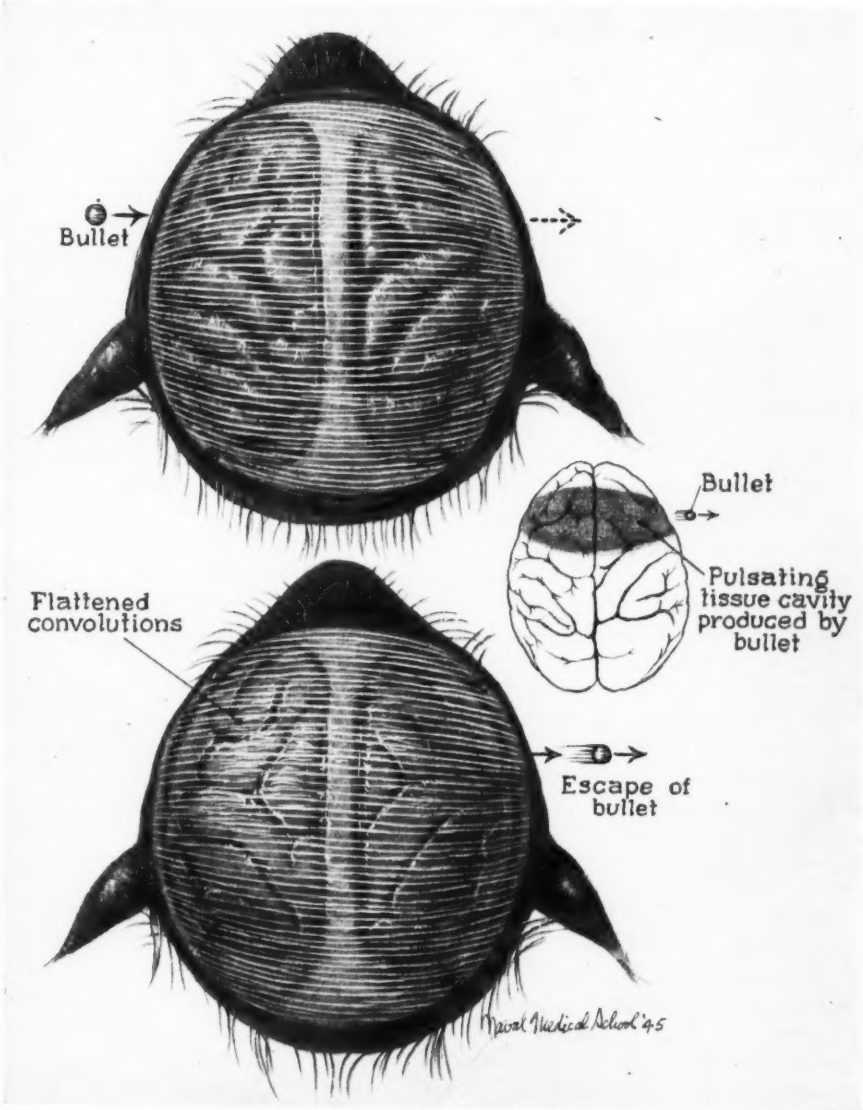

FIG. 7. Gunshot wound of the brain. The intense flattening of the convolutions at the height of intracranial pressure is shown.

demonstrated in these experiments. It has been shown that the surface movement has a gliding rotatory quality which is modified by the site and direction of the blow, by fixation or mobility of the head and by certain other factors. For purposes of simplification rotation is considered as occurring in the horizontal, coronal and sagittal planes or combinations of these planes. Blows that cause the skull to move through a simple sagittal arc cause a pure type of brain movement in the same plane. On the other hand temporal and parietal blows cause rotation of the skull and brain through a combination of planes. In these latter blows the sagittal and horizontal convolutional glides were most pronounced whereas coronal rotation was not evident. Nevertheless, it is possible that coronal rotation did occur but was either masked by the other motion patterns or was not visible under the conditions of these experiments.

Indentation of the skull resulting from the blow was noted in some of the experiments. However there was no obvious outward bending of the opposite pole of the skull as surmised by previous investigators. Furthermore there was no grossly visible change in the cerebral circulation.

The most interesting observations of this study concern:

1. The variation in the degree of motion in different areas of the brain.
2. The relationship between the amplitude of rotation and the distribution of cerebral lesions.
3. The effect of fixation of the head at the time of the blow.
4. The tamponade effect of the cerebrospinal fluid.
5. The explanation of subdural hematoma on the basis of cerebral rotation.

Each of these points may now be considered in detail.

Rotation of the cerebral mass was maximal in the parieto-occipital region and minimal in the frontal area irrespective of the site and direction of the blow. In the pure sagittal types of rotation induced by frontal and occipital blows the convolutional motion was equal on the two sides. However, when the blows were delivered to the temporal and parietal areas of the skull the amplitude of movement in all planes was slightly greater on the side of the blow.

Since the frontal lobes are subjected to the same rotational forces as the parietal and occipital lobes, their comparatively slight motion can be explained only on their anatomical position. Free motion must be inhibited by the more rigid anterior fossa as compared to the less confining posterior portion of the supratentorial space. This restraining effect of the anterior fossa on the frontal lobes must lead to strains within the cerebral tissue. Holbourn has postulated that it is these shear strains that lead to laceration and hemorrhagic softening of the cerebral tissue. Although the temporal lobes were not visualized through the lucite calvarium it is not unlikely that similar strains occur in these regions since these lobes are similarly confined by the middle cranial fossae. On the other hand the parietal and occipital lobes are less restricted in their gliding and therefore less subject to shear strains.

These observations are in accord with the actual distribution of cerebral lesions in fatal cases of head injury. In Vance's[24] series of 512 necropsies, contrecoup lesions were generally confined to the under surface of the frontal and the temporosphenoidal lobes. Blows to posterior areas of the head usually caused damage to both frontotemporal areas. Blows to the lateral areas of the skull damaged the opposite temporosphenoidal area. However, frontal blows rarely caused contrecoup lesions. Similarly, Le Count and Apfelbach[16] noted the rarity of occipital lobe injury with trauma to the frontal region.

In Courville's[5] series of 206 consecutive cases of fatal head injuries, the sites of cerebral damage were temporal lobe in 145 cases and frontal lobe in 84. Injuries to the occipital lobes were rare unless by direct contusion from a depressed fracture.

Fixation of the head at the time of the blow resulted in a considerable diminution of the rotatory motion. Apparently under the conditions of fixation the flinging movement imparted to the brain by the moving skull does not occur. Because of this, contrecoup lesions should be unusual under these conditions. This is similarly supported by the experiences of Vance,[24] Le Count and Apfelbach,[16] Munro,[19] Denny-Brown and Russell,[7] Gurdjian and Webster.[14] Vance was unable to demonstrate contrecoup lesions at autopsy in the brains of 40 individuals whose heads were crushed by an automobile on a roadway. Le Count and Apfelbach state that when the cranial bones are broken with the head in a fixed position contrecoup damage is reduced to a minimum. Munro notes that contrecoup lesions occur only when the head is struck and arrested while in motion. In their experiments on cerebral concussion Denny-Brown and Russell, and Gurdjian and Webster noted a greater degree of cerebral damage when the head was freely movable, rather than fixed, at the time of impact.

The cushioning of the brain by the cerebrospinal fluid was demonstrated in the experiment in which blows of equal force were delivered before and after the withdrawal of the fluid. Under the conditions of reduced intracranial pressure the amplitude of convolutional glide was considerably greater. This point probably has no clinical significance other than to demonstrate the supportive role of the cerebrospinal fluid. However, this finding encourages further investigation of the relationship between brain movement and concussion. If concussion is at all related to brain rotation it should be more easily obtainable under conditions of reduced intracranial pressure.

Finally, the pathogenesis of the subdural hematoma may be considered. It has long been taught that the subdural bleeding is due to the tearing of cortical veins from the sagittal sinus by the displacement of the brain. Although the actual tearing of the veins was not visualized, massive subdural ("sublucite") venous bleeding occurred following parietal blows in two animals. The bleeding occurred from parietal veins in the region where convolutional glide was greatest. This experimental finding is in harmony with the location of subdural hematomas as found in clinical practice.

SUMMARY AND CONCLUSIONS

1. High-speed cinematography has been used to observe the effects of subconcussive blows and gunshot wounds of the head on the brains of macaque monkeys fitted with lucite calvaria.

2. Blows on the head cause swirling, rotatory movements of the brain within the cranial cavity.

3. The patterns of convolutional motion are determined by the direction in which the head is displaced by the blow.

4. Midline frontal and occipital blows displace the head through a sagittal arc and produce convolutional gliding through the same arc.

5. In contrast, temporal and parietal blows rotate the head through a combination of sagittal, horizontal and coronal planes and cause a complicated type of brain movement in which convolutional glide in the sagittal and horizontal planes is striking. Coronal rotation of the brain was not visualized distinctly.

We are indebted to the staffs of the Audio-Visual Branch of the Bureau of Medicine and Surgery and of the Photographic Science Laboratory, U. S. Naval Air Station, Anacostia, D. C. for their excellent cooperation in the cinematographic studies. The cooperation of Professors E. N. Harvey and E. G. Butler and their colleagues in the Department of Biology, Princeton University, was invaluable in the study of gunshot wounds of the head. J. G. Wisda, PhM1c, V6, U.S.N.R., rendered valuable technical assistance in all of the experiments.

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THE SURGICAL TREATMENT OF HEMIFACIAL SPASM*

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HEMIFACIAL spasm, or organic spasmodic facial tic, produces paroxysmal contraction of the facial muscles on one side of the face, most noticeable around the eye, which accounts for the term "blepharospasm" being applied to the less severe syndromes. Except for the fact that a motor nerve is involved, many of the features of the affliction suggest that it is related to trigeminal neuralgia. The average age of the patients is about 45 years, and the paroxysms seem to be precipitated by eating, talking, voluntary facial movements, psychic tension, and by strong sunlight. The twitching consists of rapid multiple contractions of varying numbers of muscle fibres without any constant distribution and is most noticeable in the closure of the eye. The rest of the face, and even the platysma, is involved to some extent in most cases. The rapidity and location of the spasm suggest fibrillation rather than any coordinated or purposeful movement. It occurs during sleep.

Although psychic factors seem to increase or precipitate the paroxysms at times, there should be no difficulty in differentiating the condition from functional tic, which can be voluntarily imitated. "Paraspasm faciale," or median facial spasm, produces a slower, bilaterally symmetrical contraction and the tongue and pharynx are also involved. This condition is obviously supranuclear in origin and may be a postencephalitic or post-traumatic sequela. Great disability is produced, since the patient may close his eyes while driving a car or be placed in a similarly dangerous or embarrassing position. The methods of treatment to be outlined for hemifacial spasm are almost equally effective in this condition.

The cause of hemifacial spasm is unknown, but the history of direct trauma to the facial nerve was obtained in several cases. The condition has been observed following Bell's palsy. The lesion, according to the best evidence, lies in the nucleus of the seventh nerve. Of 663 patients examined at the Mayo Clinic for abnormal facial movements, 106 had hemifacial spasm.[2]

Because there is no pain, there has been a tendency to minimize the suffering of these people. Women, particularly, become conscious of their appearance and dread the paroxysms precipitated by psychic factors; some have complained that the spasm is often interpreted as a wink. The interference with reading, driving a car, and types of work that require accurate eyesight is a more practical disadvantage caused by the affliction.

Psychiatric and medical treatment are ineffective, although many methods have been recommended. The number alone is sufficient to indicate

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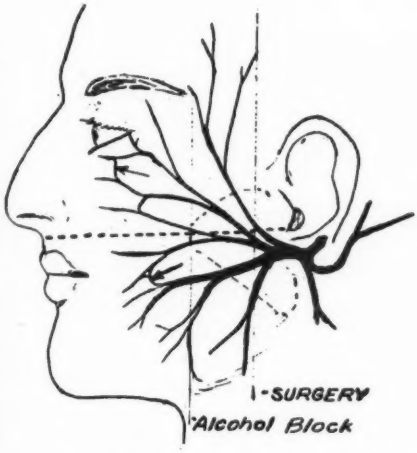
that none was successful. Ehni and Woltman[2] reported that only 5 patients appeared to be cured without surgery and 3 of these had probably only remissions. They felt that spinofacial anastomosis produced a satisfactory result in 3 cases. Coleman[1] divided and resutured the upper major division of the facial nerve just anterior to the ear. He also reported the results of hypoglossofacial anastomosis. With one exception, all methods of surgery relieved spasm only at the price of paralysis, loss of facial expression, or inevitable recurrence. Anastomosis sacrifices facial expression. German[3] re-



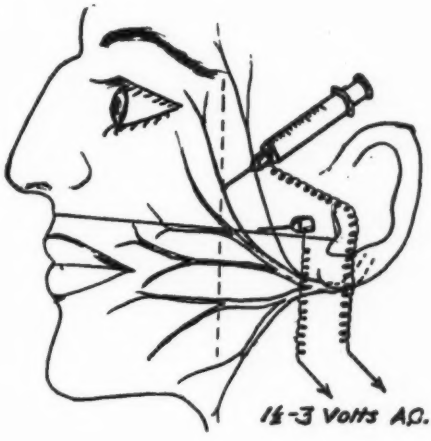


FIG. 1 (*left*). Surface anatomy of the facial nerve.
FIG. 2 (*right*). Technique of alcohol block of divisions of facial nerve to obtain weakening rather than paralysis.

ported the treatment in 5 cases by partial section of the several divisions of the facial nerve anterior to the parotid gland. Alcohol injection has never been very successful because injection at the stylomastoid foramen caused a complete, though not permanent, paralysis and injections of smaller divisions were too inaccurate to produce any uniform success. Injection cannot result in permanent relief.

The persistence of these people in their search for help and their gratitude when even minor improvements occurred led to an improvement in alcohol injection. Their willingness to submit to repeated injections made us search for a surgical technique that has fewer disadvantages than previous methods. A modification of German's method was used for surgery on the nerve. The key to both methods is the ability to locate the nerve and nerve branches by electrical current and to gauge the amount of weakness produced in the various muscle groups so that no noticeable paralysis is produced. In both methods the orbicularis oculi is usually left with sufficient strength to close the eye, yet so weak that twitchings are absent or not noticeable and the

unaffected levator easily holds the eye open. Fig. 1 illustrates diagrammatically the average anatomy of the facial nerve. The position of the parotid duct level is indicated by the dotted line extending from half way between the bridge of the nose and the lip to the external auditory canal. The parotid gland is also outlined. Two vertical lines indicate the planes along which alcohol block or surgery is done. The line for surgery is not the line for incision. Arrows indicate anastomotic connections.

ALCOHOL BLOCK

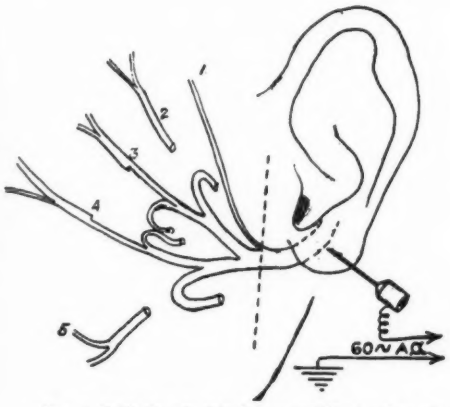
Injections are made along a vertical line just anterior to the parotid gland. This line is about $\frac{1}{4}$ inch posterior to the lateral border of the orbit. The parotid duct is palpated and a hypodermic needle is inserted just under the skin, parallel to and above the duct. This needle acts as one electrode and serves to indicate the position of the parotid duct so that no alcohol will be injected into it. One wire of the stimulating current is attached to the needle; the other side of the current is connected to a second needle which is attached to a hypodermic syringe containing 1 per cent novocaine. The needle is then introduced first at about the level of the zygoma along the injection line, and a continuous wheal of novocaine is produced just under the skin down to the level of the upper border of the mandible. The needle is then introduced again over the zygoma, but to greater depth, and 2 volts of 60 cycle alternating current is turned on and, while observing the face and with repeated attempts, the needle can be adjusted so that its tip actually touches the branch to the frontalis. This branch is not injected since it seldom takes part in the pathological process. The needle is then introduced about $\frac{1}{4}$ inch lower and the next lowest division is found (Fig. 2). One cc. of novocaine is injected; then the syringe is carefully removed from the needle and a second syringe of 95 per cent alcohol is connected and $\frac{1}{2}$ cc. injected. The contraction caused by the current will subside immediately. The same procedure is followed in the succeeding lower branches, but care is taken not to inject alcohol close to the parotid duct. After each injection the patient is asked to close his eye as tightly as possible. As a rule, there is no noticeable change after the first two injections; but on the third or fourth, a marked weakness of closure will be noticed. Along the line of injection one may find 5 to 8 branches, and the injections are continued downward until the desired result is produced. The contractions will have largely disappeared, yet the patient can raise his eyebrow, wrinkle his forehead, smile fairly well, and close his eye weakly on the affected side. The situation should be re-examined in 3 days to determine the need, if any, for injection of an additional branch or two. Except for some swelling of the face there is little reaction, and hospitalization is not necessary. In several days the amount of weakness is always less than immediately following injection.

OPERATIVE TECHNIQUE

With the patient under a general anesthetic the field is so draped that the face can be partially uncovered for observation during actual section of the facial nerve. A 2-inch vertical incision is made just anterior to the ear, extending downward from a point just above the level of the external auditory meatus (Fig. 3). If the incision is placed in a wrinkle, the scar will be practically invisible. The incision is extended partly through the parotid gland, and at this point the exact position of the facial nerve can be determined by gently passing through the gland a needle carrying $1\frac{1}{2}$ volts alternating current. By careful dissection, the nerve is followed anteriorly until it has separated into at least 4 divisions. Arising from the upper division a fine branch to the frontalis can be isolated; it is important to preserve this if possible. The separate divisions are then stimulated to determine their destination. Partial section of some divisions is done first by dividing them longitudinally with a pointed scalpel and cutting anteriorly the part to be turned back. Usually 1 or 2 divisions will be completely divided and turned back to prevent regeneration. It is not felt that the loose sutures used by German are

necessary, as the free ends turned back quickly become adherent to surrounding tissue due to fibrin formation. After each section, the whole nerve is stimulated by a needle which is kept in place with its point at the stylomastoid foramen (Fig. 3). By repeated observation the results of the serial divisions can be checked. As a rule, the amount of current for this part of the procedure should be enough to produce about half of the full facial contraction.

A typical procedure will begin by locating and sparing the branch to the frontalis. The 2nd (zygomatic) branch may then be completely sectioned. The lowest branch (cervical) is then sectioned. Testing electrically may show still too much strength and the lower half of the 3rd division, and possibly the upper half of the 4th division, is separated and turned back. At this point, spontaneous twitchings which often persist during anesthesia have probably stopped. Stimulation of the nerve at the stylomastoid foramen should cause the eye barely to



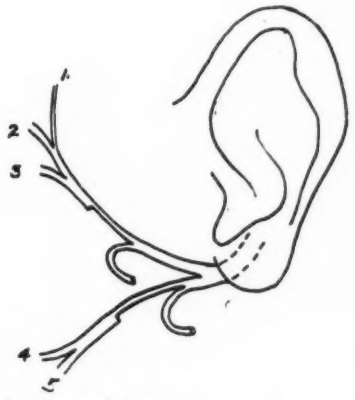


FIG. 3 (*left*). Surgical incision and illustration of type of division carried out.
FIG. 4 (*right*). Suggested division of nerve where only two branches are found in posterior parotid.

close, and should cause good movement of the cheek and forehead. Closure is done with fine cotton, including one layer just under the skin surface. This prevents widening of the scar. Occasionally the nerve does not separate into more than 2 divisions until it is well forward, in which case a division such as is indicated in Fig. 4 seems to be preferable to running the risk of damaging the larger ducts in the anterior part of the gland.

RESULTS OF ALCOHOL BLOCK AND SURGERY

In the past 3 years, 22 patients have been seen with this condition. Twelve were women and 10 were men. The average age was 46 years; only 2 cases occurred before 30. Twenty-four alcohol blocks were done on a total of 14 patients. Ten operations were done on 8 patients. In one of these the spasm was bilateral, and in another a second operation was done due to incompleteness of the first. The results of alcohol block were good, but lasted only 4 to 6 months, explaining the need for repetition. So far, only one of the patients operated upon has had any appreciable return of spasm. Two patients had a little difficulty in closing the eye, but were satisfied with the result. In no case was much facial expression lost on the side of the lesion. There have been no complications from either alcohol block or surgery, except excessive watering of the eye where closure was not complete. One of

these two cases has cleared up and the other has improved greatly. Dissection of the posterior part of the parotid gland produced no untoward effect.

SUMMARY AND CONCLUSIONS

1. Hemifacial spasm is an organic contraction of the muscles of one side of the face, probably due to irritation or hyperexcitability of the facial nucleus.

2. No medical treatment is effective.

3. This condition has been treated in the past by complete or partial section of the nerve, alcohol block, and by anastomosis, all of which formerly led to paralysis, loss of facial expression or recurrence.

4. Methods of electrical localization and estimation are presented which make possible an accurate selective partial interruption of the facial nerve divisions by alcohol or surgery. Preservation of facial expression and ability to close the eye are made possible, while spasm is practically or completely eliminated.

5. The results of treatment are summarized in 22 cases.

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CYSTIC TERATOMAS AND TERATOID TUMORS OF THE CENTRAL NERVOUS SYSTEM IN INFANCY AND CHILDHOOD

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THE teratomas and teratoid tumors of the central nervous system have been the subjects of numerous publications.[1,2,3,6] Most of these have been single case reports or systematic studies of the literature. The tumors have come, therefore, to be regarded as curiosities unlikely to be encountered by the neurosurgeon or to be available for detailed histological comparisons. Furthermore, the wide variations in structure and clinical behavior have made it difficult to draw conclusions in regard to treatment and prognosis from one or a few cases. At The Children's Hospital, there have been 15 neoplasms of this group in 20 years.* With a series as large as the present one, it is possible to survey a considerable range of histological structure and of clinical behavior as well as to evaluate different forms of surgical management.

Since certain tumors of the central nervous system have much histological resemblance to the group under discussion but present entirely different surgical problems, it has been necessary to set arbitrary limits to the series. Craniopharyngiomas and sacrococcygeal teratomas have been excluded, as have chordomas. We have included the dermoid cysts, however, because they present neurosurgical problems similar to those of the cystic teratomas and because it is our belief that they belong histologically and developmentally to the same group as the tumors containing more varied organoid elements.

The 15 teratomas and teratoid tumors were found among 231 neoplasms of the central nervous system operated upon at The Children's Hospital (6.5 per cent). Eight of the tumors were intracranial (4.1 per cent of all intracranial tumors at this hospital) and seven were intraspinal (18 per cent of all intraspinal tumors). In interpreting these statistics, it should be borne in mind that The Children's Hospital has no patients over 15 years of age. The pathological features of the intracranial and intraspinal teratomas and teratoid tumors were identical and will be discussed together. From the standpoint of surgical management, the problems were somewhat different; the clinical features of the two groups will accordingly be considered separately.

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* Three more patients with intracranial cystic teratomas have been added to the series since the preparation of this report, making a total of 18.

TABLE 1

Intracranial cases

Case No.	Age at Onset	Sex	Clinical Data	Location	Associated Anomalies	Operation	Histology	Result
1 C.W.	10–8/12 years	M	Diplopia on looking to left. Increased intracranial pressure.	Third ventricle	None	1. Removal of cystic tumor from third ventricle. 2. Exposure of inoperable residual.	Stratified squamous epithelium, pseudostratified columnar epithelium, gland-like structures with various epithelia, smooth muscle, peripheral nerve, collagenous tissue.	Good immediate result; recurrence after 5 months. Death 10 months after operation.
2 R.T.	8 days	M	Gradual increase in size of head.	Frontoparietal	None	Removal of cysts.	Cuboidal epithelium, pseudostratified columnar epithelium, ? choroid plexus, atypical blood vessels, collagenous tissue.	Temporary improvement. Living 18 months after operation.
3 R.B.	10–10/12 years	F	Not related to teratoma.	Choroid plexus	Defects septum pellucidum	Removal intraspinal meningioma.	Whorls of arachnoid cells with psammomas, stratified squamous epithelium, bone with marrow, nerve trunks, smooth muscle, collagenous tissue.	Incidental finding at autopsy.
4 A.M.	10–4/12 years	M	Vomiting 3 months. "Hypertrophic gastritis."	Cerebellar	None	Incomplete removal 4th ventricle tumor.	Atypical nerve cells with mitotic figures, striated muscle with rhabdomyosarcomatous changes, collagenous tissue.	Death 12 months after operation.
5 C.G.	6 months	F	Hemiplegia, coma, block.	Cerebellar	None	Removal mid-line posterior fossa tumor.	Hair, hair follicles, collagenous tissue; inflammatory and foreign body reaction.	Living 9 years after operation. ? Slight mental retardation.
6 R.P.	5–10/12 years	M	Drowsiness, headache, vomiting.	Cerebellar	None	Evacuation of cyst.	Stratified squamous epithelium, edematous collagenous tissue.	Death 11 days postoperative.
7 R.W.	3 years	F	Vomiting, increased pressure	Fourth ventricle	None	Posterior fossa exploration. Removal of cyst.	Stratified squamous epithelium, collagenous tissue.	Symptom-free 4 years postoperative.
8 M.W.	Birth	F	Hydrocephalus	Frontal bilateral	None	Right frontal bone flap. Partial removal large tumor.	Serous glands, mucous glands, voluntary muscle, fat, cartilage, collagenous tissue, pseudostratified columnar epithelium, lymphatic vessels, Pacinian corpuscles.	Died 3 days postoperative.

TABLE 2

Intraspinal cases

Case No.	Age at Onset	Sex	Clinical Data	Location	Associated Anomalies	Operation	Histology	Result
9 C.A.	4 months	F	Slow development.	Cervical	None	Laminectomy. Removal cystic tumor from cervical cord.	Stratified squamous epithelium, transitional epithelium, fat, collagenous tissue, hair follicles, sweat glands, ? ependyma, nervous tissue.	No recurrence 3½ years postoperative.
10* C.S.	2–4/12 years	F	Neck pain, sudden quadriplegia.	Cervical	Occult spina bifida over lesion.	Laminectomy. Evacuation of cyst. Removal solid tumor.	Pseudostratified ciliated epithelium, mucous glands, cartilage, alveolar spaces lined by cuboidal epithelium, fat, nerve fibers, collagenous tissue.	Living with no residual changes 10 years after operation.
11 R.M.	5 years	M	Back pain, paralysis lower extremities, C.S.F. block.	Lumbar	None	Laminectomy. Incomplete removal cystic tumor.	Stratified squamous epithelium, sebaceous glands, mucous glands, pseudostratified columnar epithelium, fat, collagenous tissue.	Living with few residual changes 4 years after operation.
12* J.W.	Birth	M	Weakness extremities, pneumonia.	Cervicodorsal	Wrinkled red skin and spina bifida over tumor.	Laminectomy. Incomplete removal cervical cyst.	Columnar epithelium, stratified squamous epithelium, collagenous tissue.	Death due to pulmonary infection 5 months postoperative.
13 P.C.	6–8/12 years	M	Vomiting, abdominal pain	Dorsal	Dimple in skin, tract running into canal.	Laminectomy. Removal of cyst.	Stratified squamous epithelium, collagenous tissue, hair follicles.	Well 3½ years postoperative.
14 P.D.	1 year	F	Weakness of legs, C.S.F. block.	Lumbar	Occult spina bifida.	Laminectomy. Incomplete removal of tumor.	Nervous tissue, fat, thyroid tissue, smooth muscle, collagenous tissue.	Well 3½ years postoperative.
15† D.S.	2 years	M	Back pain, C.S.F. block.	Lumbar	Occult spina bifida.	Laminectomy (2). Removal cystic tumor.	Columnar ciliated epithelium, mucous glands, collagenous tissue.	Well 19 years postoperative.

* Previously reported by Ingraham.[4]

† Previously reported by Ingraham[4] and Kubie and Fulton.[5]

PATHOLOGICAL CONSIDERATIONS

Tables 1 and 2 show the incidence in which mesodermal, entodermal and ectodermal derivatives were represented among the 15 tumors. The simplest contained only stratified squamous epithelium undergoing keratinization and its accompanying stroma of collagenous tissue. In the series, there were tumors varying from these dermoids to neoplasms composed of a great variety of tissues developing from all three germ layers.

As seen grossly, whether at operation or at autopsy, the lesions had much more in common than in histology. A striking characteristic of the solid portions of the lesions in the gross was the texture of the tissue. It was firm and grayish-white, at times with areas discolored yellow by previous hemorrhage. This was an indication that the tumors contained sufficient collagenous connective tissue to determine the texture of their solid portions and was a great assistance to the surgeon in differentiating them from tumors of the glioma group. In the case of neoplasms made up only of keratinizing epithelium and collagenous tissue, the latter seemed best interpreted as a stromal response. When the tumor contained bone, cartilage or other mesodermal derivatives as well as epithelium, it was impossible to tell how much of the collagenous tissue was an element of the tumor itself and how much was stroma, incited to proliferation by other elements.

Some of the teratomas were sharply demarcated from the adjacent nervous structures and could be readily dissected away from them. Others, and especially in the intraspinal group, blended so closely with the brain or spinal cord that no plane of cleavage could be identified. Histologically, the blending was the result of a mixture of reactive gliosis with the connective tissue of the tumor. In one instance (Case 4), the tissue elements were themselves invasive, the neoplasm being composed of a mixture of neuroblastoma and rhabdomyosarcoma. Special features of this and certain other tumors are discussed in the individual case reports.

In all instances, some part of the tumor was cystic. The cysts were usually multiple (Figs. 2 and 12) and varied in size from microscopic structures (Case 3) to huge, tense masses of fluid comprising a large part of the cranial contents (Case 2). The character of the fluid varied greatly, not only from tumor to tumor but also occasionally from cyst to cyst within a single tumor. In some instances (e.g., Case 2), the fluid was pale yellow and clotted. If a small quantity of such fluid was obtained from a cyst or by aspiration through a needle inserted through a suture or burr hole, it would be indistinguishable from that of a gliomatous cyst or late subdural hematoma. When a cyst of a teratoma contained this type of fluid, it was usually present in much larger amounts than would be expected in either of the two other conditions. At times, the fluid in a cystic teratoma was deep yellow and clotted, like the first type. Here again, the differentiation from the fluid of certain gliomatous cysts or of one stage of subdural hematoma could not be made on the character of the fluid alone. More suggestive of the diagnosis of cystic teratoma or teratoid tumor was a cloudy, whitish fluid with a

yellow tinge. This was not cloudy because of blood but because it contained cells. The fluid was not purulent, and few leukocytes could be demonstrated. It was cloudy in most instances because of keratinized cells cast off by stratified squamous epithelium lining the cyst. This is the type expected in dermoid cysts but may also occur in any teratoma in which there is a cyst lined by keratinizing epithelium. In Case 15, the cells in the fluid were columnar and had cilia, as described by Kubie and Fulton.[5] In still other teratomas, a chocolate-colored, opaque, stringy, gelatinous fluid was obtained; at times, this had a reddish tinge. While it is possible that fluid of such character might be found in a necrotic glioblastoma multiforme, we have seen it only in instances of cystic teratoma or teratoid tumor.

At least two factors help to determine the character of the fluid in these cysts. One is the nature of the cells which form the lining—whether or not they produce keratinized epithelium or other desquamated cells, whether or not they secrete mucin, whether or not they are necrotic. The other factor is the presence or absence of hemorrhage. If there has been recent hemorrhage, a reddish tinge may be expected; if old, the fluid will be yellow or brown. Because of the variability in the character of the fluid in cystic teratomas and teratoid tumors, it is usually unwise to depend wholly on the gross appearance of the fluid when considering the diagnosis in the operating room. A stained smear of the fluid will often show keratinized cells, ciliated epithelium or other elements which make the diagnosis certain at an early stage in the operative procedure. The presence of hair within the cyst when opened at operation, of course, establishes the nature of the lesion without further procedure, but is uncommon (Case 5).

We do not feel that our study of the 15 tumors in the present series contributes anything toward an understanding of the histogenesis of the condition. Neoplasms of this group, in the central nervous system and elsewhere, have long been regarded as the result of belated and abnormal proliferation of pluri- or totipotential cells misplaced in early embryonic life. It is as difficult to find an alternative for this view as it is to obtain direct evidence bearing on its validity. Perhaps one reason why the dermoids of the central nervous system have been set apart in most publications from the other tumors that have here been grouped together is because they contain tissues derived only from the ectoderm along with a connective-tissue stroma, developed in response to the presence of the ectodermal structures. Since the nervous system adjacent to the dermoid is also ectodermal, there has been the suggestion that the factors inducing the development of the tumor occur much later in embryonic, or even in postnatal, life. So far as can be determined from the present series, the dermoid forms one end of a scale of increasing complexity, with the highly varied tridermal tumors at the other.

Vonderahe and Niemer[8] have published an interesting study of the relationship of intracranial lipomas to teratomas and propose a correlation between the histology of the tumors and the stage of embryonal development at which the tumor originated. They say, "Lipomas developing some time

after gastrulation contain only adipose tissue; others, developing shortly after gastrulation, possess more highly differentiated mesodermal derivatives such as bone; still others, developing before or during gastrulation, possess neuroectodermal as well as mesodermal elements. There is thus a transition from simple lipoma to the more complex teratoid tumors and teratomas." In the same way, the tumors in this series seem to vary from one another only quantitatively, and in the embryonic potentialities of their cells of origin. Russell[7] has shown that certain pinealomas are actually atypical teratomas but can be proved so only on detailed histological study. The histological possibilities of the teratoma group appear to be more varied, the more they become understood.

CLINICAL ASPECTS—INTRACRANIAL GROUP

From the clinical point of view, the character of the symptoms and the problems of differential diagnosis of intracranial teratomas and teratoid tumors were those of any space-occupying lesion in that location. There were local effects, depending on the site of the tumor, and pressure effects, resulting from the increase in volume of the cranial contents. Nothing was encountered that would suggest the pathological diagnosis preoperatively, unless an associated congenital anomaly was present. This occurred occasionally in the intracranial group (Case 5) but more often in the intraspinal group. The presence of a congenital anomaly in a patient with signs and symptoms of a space-occupying cerebral lesion does not establish the diagnosis of teratoma or teratoid tumor, but makes the chances of finding this lesion somewhat greater than in its absence. It would be unusual to find a glioma in a patient with a skull defect or superficial malformation of the head, but hemangiomas or similar tumors might well be encountered.

On the whole, the symptoms of the tumors in the series developed slowly (e.g., Case 5) not only because the neoplasms were slow in growth but also because children tolerate increased intracranial pressure better than adults. There was no evidence that the cysts filled suddenly with fluid. While there was the possibility of a large hemorrhage into a pre-existing cyst with consequent enlargement of the volume of the tumor, this was uncommon. Case 4 was an exception, in that symptoms developed rapidly. This was the result of the histological character of the tumor, which was invasive and contained numerous mitotic figures.

Roentgenograms of the skull might possibly show calcified areas suggesting the presence of bone or other organoid structures in tumors of this group. In none of the patients in this series was it possible to make a definite diagnosis of the type of lesion from the roentgenograms. There was evidence of increased intracranial pressure and, in some, internal hydrocephalus was present. Ventriculograms or encephalograms demonstrated filling defects indicating the size and location of the tumors.

The intracranial cystic teratomas and teratoid tumors varied a great deal from the operative point of view. Some were clearly demarcated by a con-

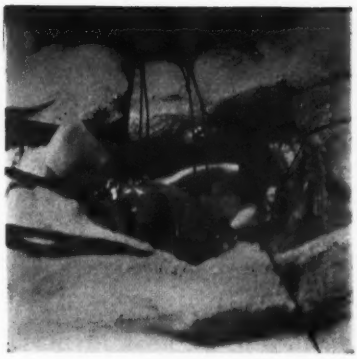

FIG. 1. Case 9. Removal of an intramedullary cystic mass from the cervical cord.

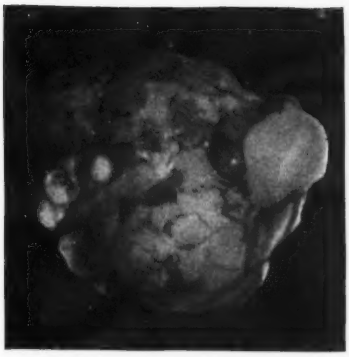

FIG. 2. Case 1. A cystic teratoma after removal from the third ventricle. There are several cysts of different sizes separated by dense tissue.

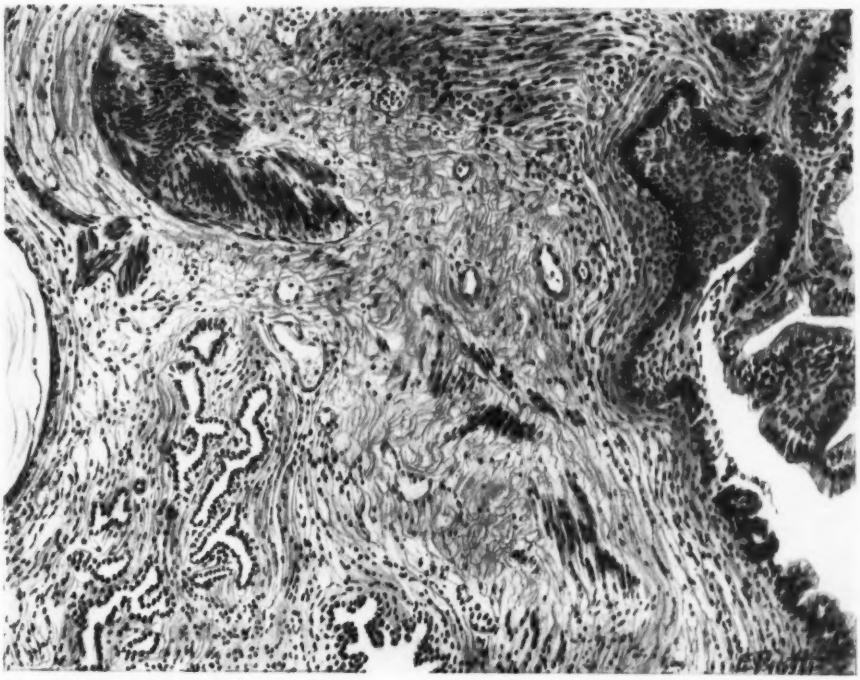

FIG. 3. Case 1. The histological appearance of an area from the tumor in Fig. 2. A cyst lined by keratinizing epithelium and another lined partly by columnar and partly by stratified squamous epithelium, and a group of gland-like structures are separated by collagenous tissue. Phloxine-methylene blue stain, camera lucida drawing, ×125.

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nective-tissue capsule and could be readily dissected away from the adjacent nervous structures (e.g., Cases 1 and 5). On the other hand, the tumor in Case 4 was so intensely adherent to the brain substance that no plane of cleavage could be found. Most instances fell between these extremes. However, in dealing with such tumors as those in Cases 2 and 8, it was impossible to define the relation to the nervous tissue because the tumors filled a large portion of the cranial cavity. The easily separable tumors offer a good prog-

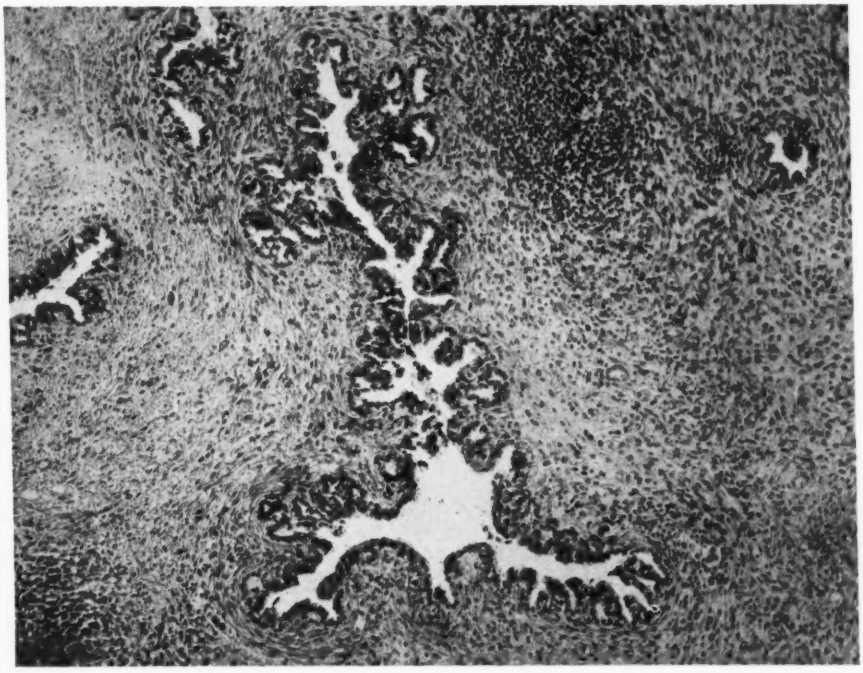

FIG. 4. Case 1. A complicated duct-like structure with many branches. Some of the cysts appeared to have originated from distension of similar cell-groups. Phloxine-methylene blue stain, ×112.

nosis. Much less can be done for the neoplasms intimately adherent to the nervous tissue, though even here much can be accomplished unless the tumor is histologically malignant or unless it is of such large size that any surgical approach is hopeless.

Of the 8 patients with intracranial teratoma or teratoid tumor, 1 is living more than five years after operation; 2 are living for periods less than five years; and 5 are dead.

The following case reports indicate the range of variation in clinical behavior and pathological findings in this group of tumors.

Abstract Case 1, C.R.W. (296488). This patient was referred to the hospital at the age of 10 11/12 years with a history of frontal headaches associated with sinusitis and ptosis of the left eyelid. For 3 months there had been double vision on looking to the left. The positive findings on physical examination were ptosis of left eyelid, papilledema with retinal hemorrhages, and marked tachycardia with a loud systolic murmur. There was moderate leukocytosis and elevation of the sedimentation rate. Roentgenograms showed a small area of calcification in the pineal region slightly to the right of the midline. Under avertin-ether anesthesia exploration was attempted but there was a marked fall in blood pressure before the scalp incision had been completed. He was very soon in a state of severe shock, operation was interrupted

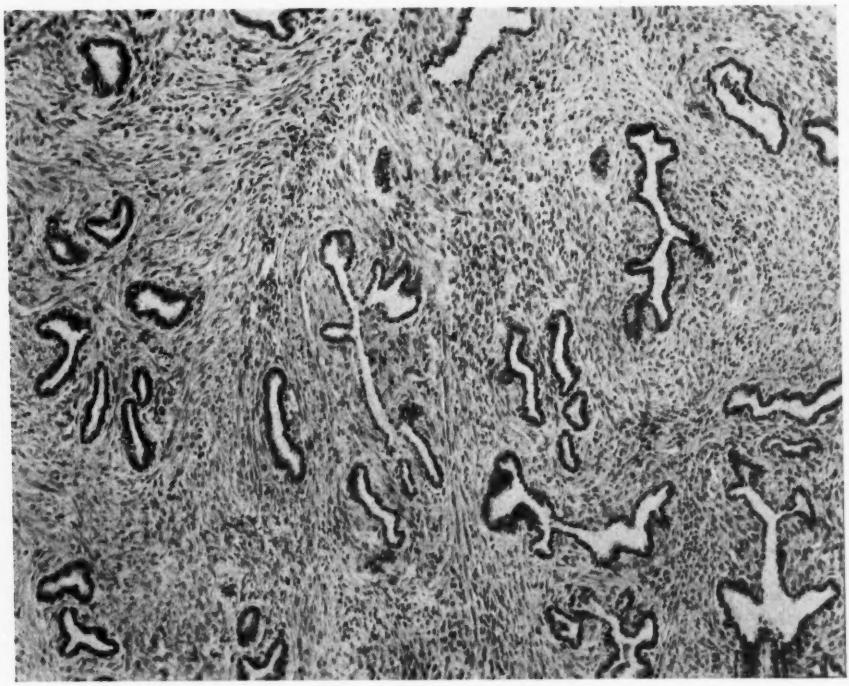

FIG. 5. Case 1. A portion of the tumor composed of glands varying in size and outline. They are not sufficiently differentiated to make identification of their origin possible. Phloxine-methylene blue stain, ×106.

and the blood pressure was restored to normal levels only after the most heroic measures including the intravenous administration of large amounts of eschatin. At a later date he was given eschatin preoperatively as well as throughout the procedure and it was possible to turn down the bone flap and make a subtemporal decompression. Operation was again interrupted as signs of shock appeared. At a third session it was possible to amputate the right occipital pole, traverse the corpus callosum and remove an intact partially cystic tumor from the 3rd ventricle (Fig. 2). It is interesting to note that the pulse rate immediately became slower, blood pressure stabilized, and there was no suggestion of shock after the removal of the tumor. It was assumed at this time that the tumor had been entirely removed and the patient was sent home in good condition 3 weeks after operation. He returned to school and was symptom-

free for 5 months, at the end of which time he complained of headache and he was readmitted to the hospital with high intracranial pressure. Ventriculography revealed the presence of a large mass in the 3rd ventricle which was found to be continuous with the adjacent brain tissue and quite inoperable. It seemed evident at this time that the large mass previously removed was a relatively free and benign portion of a larger invasive tumor. This impression was later confirmed by autopsy. The temporary relief of block in circulation of cerebrospinal fluid

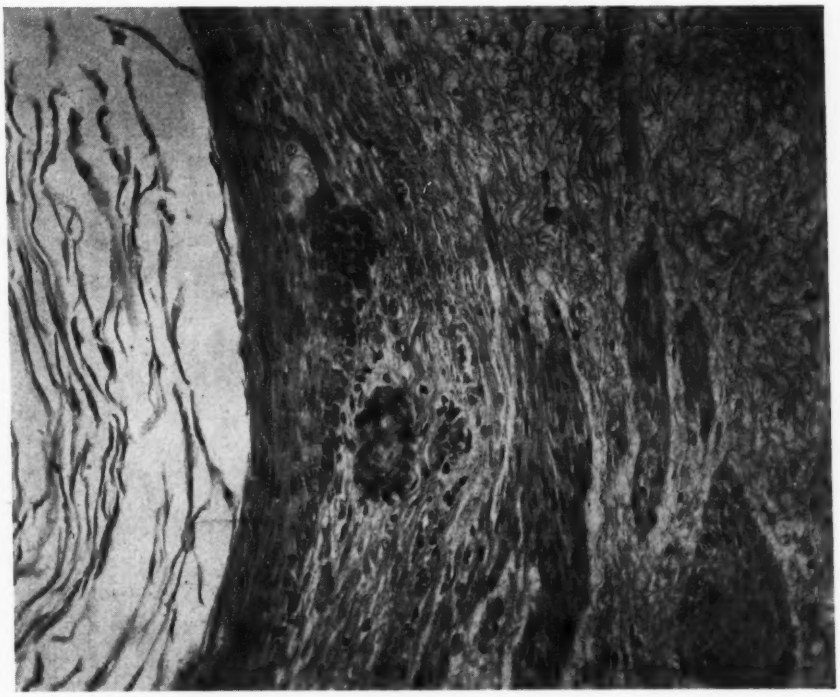

FIG. 6. Case 1. A cyst lined with stratified squamous epithelium and containing desquamated keratinized cells. The cyst is surrounded by collagenous tissue with bundles of smooth muscle concentric with the cyst lining. Phloxine-methylene blue stain, ×106.

and relief of hypothalamic pressure had been sufficient to restore the patient to a normal physiological level.

Pathological Study. The soft, grayish-pink tumor measured 3.8×3.2×2.3 cm. (Fig. 2). The cysts ranged from 0.2 to 1.5 cm. in diameter. Their walls were thin but varied in translucence. A few hard, white nodules, 0.8×0.8×0.4 cm. in size, were found on one surface of the mass. On section, the tissue had a lobulated appearance. The cysts contained clear fluid and were separated by fibrous septa, which were almost gristly in consistency.

The microscopic sections showed a variety of structures. The larger cysts were lined by stratified squamous epithelium with well marked keratinization, despite the clear appearance of the fluid. Other smaller cystic spaces consisted of duct-like structures lined by pseudostratified columnar epithelium resembling that of the respiratory tract. The epithelium was much infolded and at times showed an abrupt transition to the stratified squamous type (Fig.

3). Small glands of irregular outline were the dominant tissue element in certain areas. They were lined by low cuboidal epithelium. A few other spaces were lined by various types of epithelium, at different places resembling somewhat colonic, transitional and ependymal epithelia. The collagenous stroma contained bundles of smooth muscle and peripheral nerve. At times, the connective tissue was arranged like that in neurofibromas. Lymphocytes were present in fairly large numbers in the stroma and there were a few cells with large pale nuclei suggesting reticulum cells. No mitoses or areas of invasion were found (Figs. 3, 4, 5 and 6).

Comment. Serious physiological instability was caused by the presence of the tumor in the neighborhood of the third ventricle and pressing upon the hypothalamus. The surgery necessary for the removal of the neoplasm would probably have been impossible without the use of adrenal cortical extract. When this was given in amounts sufficiently large to restore the patient's physiology to somewhere near normal, the operative procedure could be carried out safely. The remarkably small number of successful operations on tumors of the pineal region serves to emphasize the gravity of surgery in this location when the physiological disturbances have not been corrected.

Despite the encapsulation of the tumor and its benign appearance histologically, there was evidence of recurrence five months after operation and death one month later. In the interval, the patient was in good health. From the standpoint of useful function, therefore, the quadrantic defect caused by the removal of the occipital lobe was less serious than the choking of the optic discs, which was relieved by the operative procedure.

Abstract Case 2, R.T. (290430). This infant was admitted to the hospital at the age of 8 days because of a gradual increase in the size of the head. Delivery was normal and at full term. Nothing unusual was noted immediately after birth but within a few days it was apparent that the head was growing rapidly, particularly the "back" of the head. The patient lay in a position of opisthotonos, the right side of the head down, moving the extremities vigorously. The fontanelles were large and the sutures widely separated, the left coronal more than the right. The right ventricle was tapped and pressure found to be elevated. Ventriculography showed dilatation of the ventricular system with a shift to the right. A large bone flap was turned down and a huge cystic mass was exposed (Fig. 7). The multilocular cyst appeared to fill the major portion of the left half of the cranial cavity. The cysts were evacuated and part of the wall removed but in view of the extensive damage to the brain already evident no attempt was made at complete removal.

Pathological Study. The material removed for biopsy consisted of a deep red, opaque piece of cyst wall, measuring 0.9×0.8 cm. The outer surface was smooth, while the grayer inner surface had a small brownish, nodular excrescence upon it and was traversed by several large, congested blood vessels.

Microscopically, the wall of the cyst was found to consist of coarse collagenous tissue and large endothelial-lined spaces. At a few points, the surface of the cyst was lined by a single layer of low cuboidal epithelium which immediately overlay groups of large blood vessels. In most portions of the cyst wall, however, the connective-tissue fibers formed the lining. Projecting into the cavity of the cyst were many long, fine, filamentous processes covered with epithelium similar to that of the cyst wall. Within most of the processes were distended blood vessels, being separated from the epithelium only by their endothelial layers. The picture, therefore, simulated in many respects a congested choroid plexus. A probable diagnosis of teratoid tumor was made on the basis of a cystic neoplasm with epithelial elements and an abnormal vascular tissue.

Comment. Case 2 illustrates the type of teratoid tumor for which surgery offers least. Occupying a large portion of the cranial cavity, the tumor could not be attacked radically. The most that could be done was to evacuate all accessible cysts with the hope of palliation.

Abstract Case 3, R.B. (154906). A 10 10/12 year old girl was admitted to the hospital because of weakness of the right arm of 2 weeks' duration. Physical examination showed weakness of the right leg, diminished sensation over the right forearm, right exophthalmos and

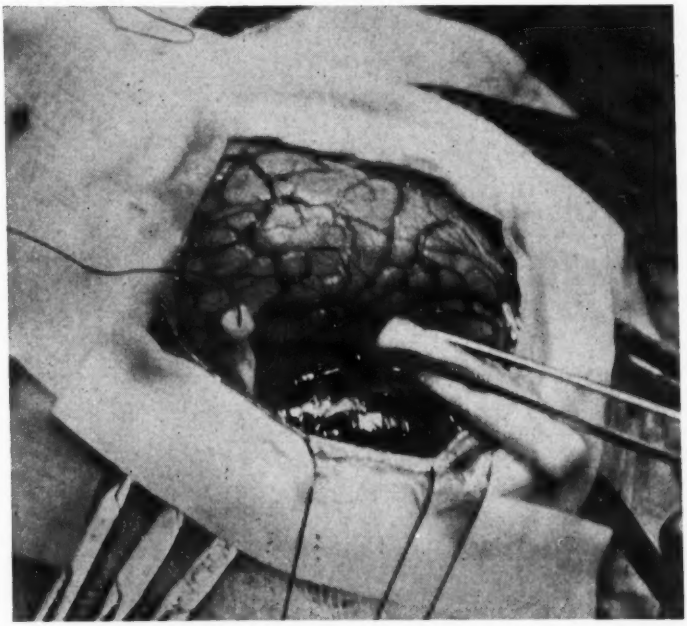

FIG. 7. Case 2. A huge cystic intracranial teratoma exposed at operation. Note the character of the cyst lining.

choroiditis. There was a *café au lait* spot on the trunk and a small cutaneous nodule in the left axillary region. This was removed and identified as a neurofibroma. Roentgenograms showed bilateral calcification of the choroid plexus. Laminectomy was done and a meningioma removed from the cervical canal. Subsequently a cerebellar exploration was done to relieve a block caused by what appeared to be a tumor of the right cerebellar hemisphere, sections of which showed gliosis but included a number of psammoma bodies. The patient died 2 years after the operation.

Pathological Study. The final autopsy diagnoses were: brain tumors, multiple; meningioma of cervical spinal cord; teratoid tumor of choroid plexus; congenital defects in septum pellucidum; sclerotic atrophy of cerebellum; syringomyelia; cutaneous neurofibroma.

The present study was concerned only with the teratoma of the choroid plexus, an incidental finding in a patient with several clinically more important lesions of the nervous system. This description is confined to the region of the lateral ventricles and choroid plexus. The lateral ventricles were slightly distorted, giving the appearance that the right basal

nuclei were pushed upward and medially, decreasing the size of the right lateral ventricle. They were not dilated. The ependymal surfaces were smooth. The choroid plexus in both lateral ventricles had a rounded, nodular appearance instead of the usual frond-like structure (Fig. 8). Between the lateral ventricles, there were multiple small defects in the septum pellucidum.

Some few portions of the choroid plexus had a normal histological appearance. The knob-like projections seen in the gross were composed of large nests of arachnoid cells arranged in whorls. There were numerous calcified psammomas within the arachnoid cell clusters. The appearance was that of a series of minute meningiomas, adjacent but usually discrete. However, there were other tissue elements near the arachnoid cell nests. A small cyst was lined

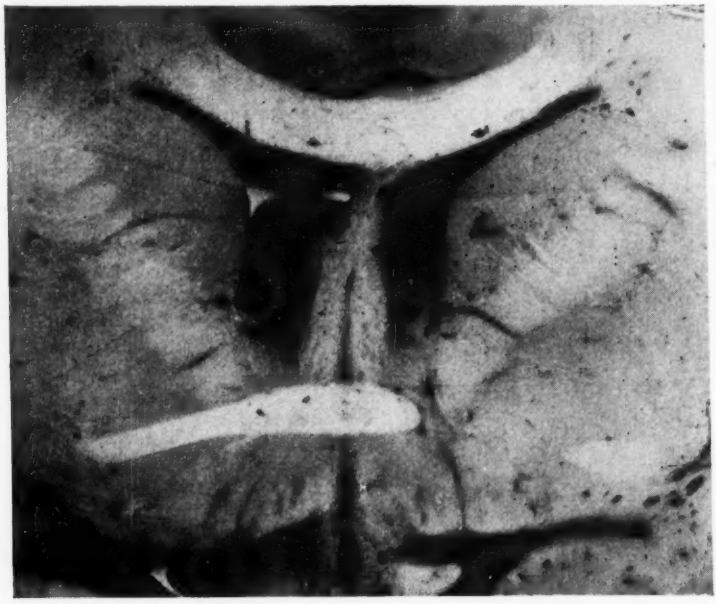

FIG. 8. Case 3. The gross appearance of a teratoid tumor of the choroid plexus. This was an incidental finding at autopsy in a patient operated upon for a meningioma of the cervical spinal cord.

by stratified squamous epithelium; its lumen contained desquamated cells which were partially keratinized (Fig. 9). At another point, a mass of bone was found. This was well calcified and had clearly defined Haversian systems. Fatty marrow was seen within the bone and small clusters of hematopoietic cells were present (Fig. 10). Several groups of myelinated nerve trunks lay within the tumor and were regarded as a part of it, since there was no evidence of invasion and since nerve trunks of this type would not be expected in the region where they were found. There was a stroma of collagenous tissue throughout the neoplasm. In spite of the resemblance of large parts of the tumor to meningioma, it was classified as a teratoid tumor because a cyst lined by stratified squamous epithelium, nerve trunks and bone with marrow were also present.

Comment. The tumor in Case 3 was an incidental finding in the midst of bewildering neuropathological abnormalities. It accounted for the calci-

fication noted roentgenologically in the region of the choroid plexus but could not be related to any of the symptoms of the patient. This was the only tumor in the series not attacked surgically. From the clinical viewpoint, it was the antithesis of Case 2, where the tumor was so large as to render radical attack impossible. The cervical meningioma in this patient was one

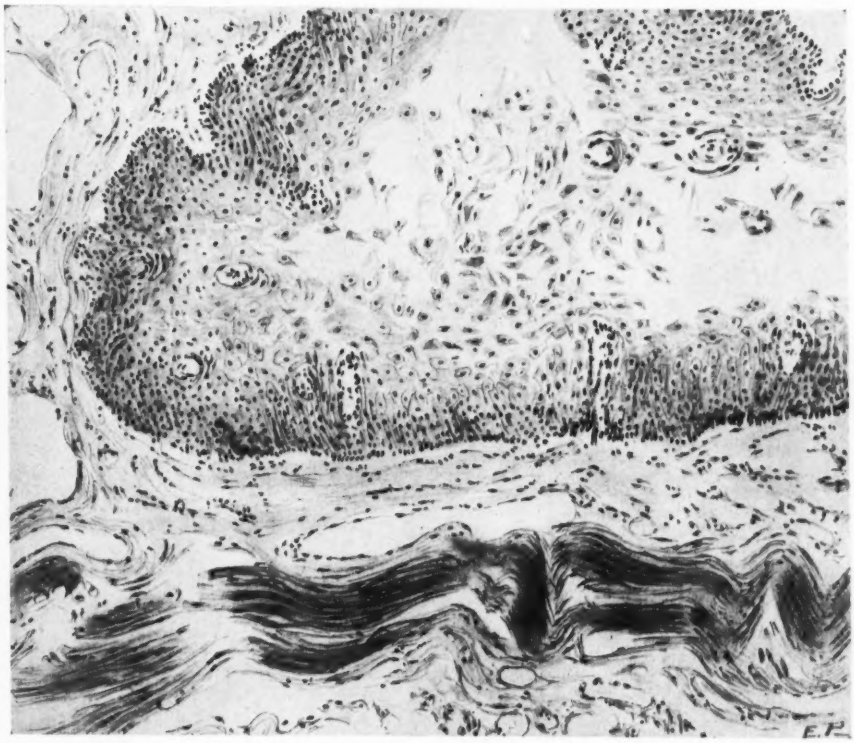

FIG. 9. Case 3. Cyst lined by stratified squamous epithelium and surrounded by smooth muscle in a teratoid tumor of the choroid plexus.

of two encountered among 231 tumors of the central nervous system at The Children's Hospital.

From the pathological point of view, the tumor in Case 3 was of considerable interest. Arachnoid villi are present normally along the septum pellucidum and hyperplasia of arachnoid villi is a fairly common finding, but the masses in this tumor achieved a degree of hyperplasia not seen under ordinary conditions. The presence of other organoid elements in association with the meningioma-like cell nests made the diagnosis of teratoid tumor necessary. No other tumor in this series had clusters of arachnoid cells among its tissue elements.

Abstract Case 4, A.M. (260086). A 10 7/12 year old boy was referred into the hospital because of vomiting spells of 3 months' duration. He vomited once or twice a day throughout this time and had lost 10 pounds. Vomiting was not associated with nausea and it was not projectile. Eyegrounds were normal. Roentgenological examination of the gastrointestinal tract showed marked prominence of the rugae interpreted as hypertrophic gastritis (Fig. 11). On the 18th hospital day he complained of double vision and shortly thereafter minimal choking of the discs could be seen. A diagnosis of midline cerebellar tumor was made and exploration carried out. A soft, reddish-gray tumor mass was exposed by incising the vermis and

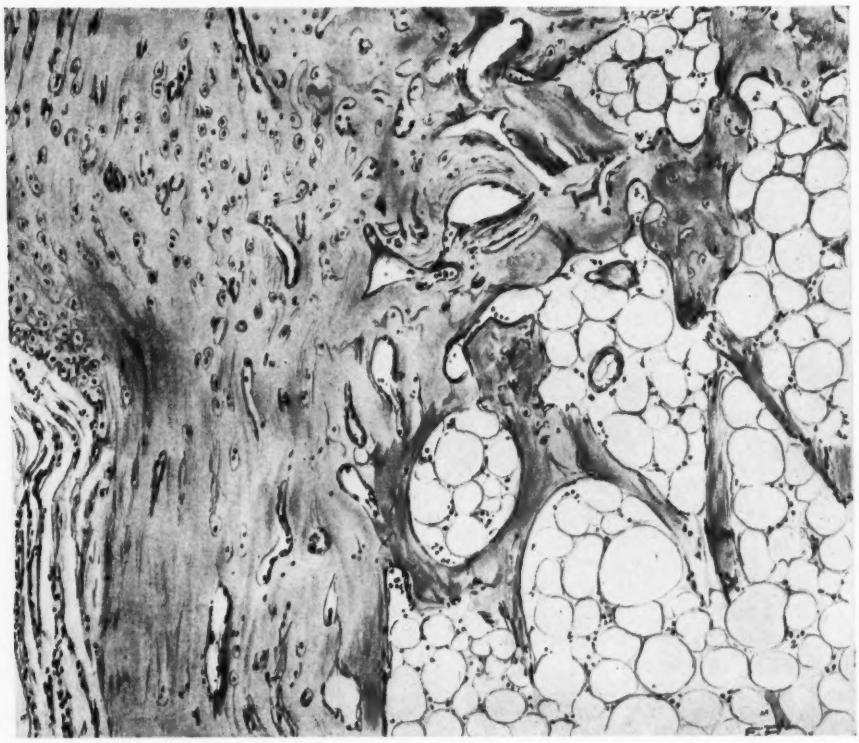

FIG. 10. Case 3. Bone with fatty marrow containing small groups of hematopoietic cells in a teratoid tumor of the choroid plexus.

the major portion of the tumor removed but the remainder was clearly continuous with the wall of the 4th ventricle. The patient made a satisfactory recovery, was given extensive radiation therapy and was free from symptoms for nearly a year. He then went downhill very rapidly and died outside the hospital.

Pathological Study. The portion of tumor removed at the first operation was composed of soft, gray fragments of tissue measuring in aggregate $2 \times 2 \times 0.5$ cm. and a thin piece of membrane, 1.2×0.5 cm.

Histologically, the tumor was made up of two types of cells. The first type had oval, somewhat vesicular nuclei closely packed with chromatin and a very small amount of cytoplasm; mitotic figures were occasionally present. Occasionally neurofibrils could be demon-

strated in these cells. The cells grew in sheets and were accompanied by a definite but scanty connective-tissue stroma. The second type of cell was found where the tumor had invaded the cerebellum and was mixed with otherwise relatively normal cerebellar tissue. Some of these cells were of medium size with darkly stained nuclei. Numerous ones were much larger—two or three times as large as Purkinje cells. The giant cells had a single nucleus surrounded by a large amount of relatively clear cytoplasm in which cross striations could occasionally be seen. In some places, there were cells with short, stubby extensions of cytoplasm containing very clearly defined cross striations. Mitoses were frequent in cells of this sort. This second type of cell was that of a malignant tumor of striated muscle. It appeared that the first type

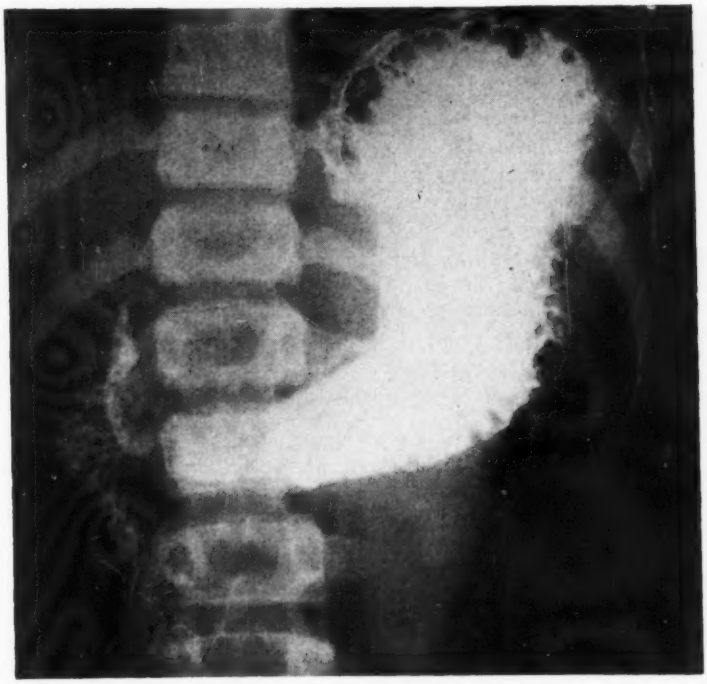

FIG. 11. Case 4. Roentgenogram showing hypertrophic gastritis in a patient with a malignant intracranial teratoid tumor. Note the greatly enlarged rugae. The "gastritis" did not change significantly after successful surgical attack on the tumor.

of cell was of neurogenic origin and completely different from the second type. The presence of two distinct types of cells in a neoplasm with a cyst led to the diagnosis of malignant teratoid tumor.

Comment. This tumor differed greatly from the other members of the series in being composed of cellular elements that were highly undifferentiated and invasive. The frozen section made during operation showed only poorly differentiated cells with many mitotic figures. Both the operative

appearance of the tumor seeding the meninges and the frozen section were suggestive of medulloblastoma. The permanent sections left no doubt as to the presence of further malignant elements and placed the lesion in the category of invasive teratoid tumors. One type of cells was the neuroblast in various stages of differentiation. The other was a striated muscle cell as seen in rhabdomyosarcoma. Both mesodermal and ectodermal elements participated in malignant growth, and no adult tissue structures were found.

The tumor in Case 4 was hopeless from the surgical standpoint, not because of large size or of inaccessible location, but because of its pattern of cellular growth. This is the second example of a malignant teratoid tumor in the series. The rarity of such lesions precludes any evaluation of the efficacy of treatment.

It is interesting to consider in this instance the relationship of this infiltrating fourth ventricle tumor to the hypertrophic gastritis. There was no vomiting during the postoperative survival period until the terminal phase. It must be assumed therefore that the prominence of the gastric rugae was the result of the long-continued vomiting before operation or was merely an incidental finding. Other children who have been vomiting for a long period of time have not ordinarily shown these changes. In an attempt to clarify the relationship, a second G.I. series was done six months after operation when the boy was perfectly well. At that time the findings were exactly the same as they were preoperatively.

Abstract Case 5, C.A.G. (208099). This patient was admitted in coma following the sudden development of paralysis of the left arm and leg. Examination showed the head to be enlarged ($19\frac{1}{4}$ in. circumference), fontanelle tense, and just below the external occipital protuberance there was a small red area which became dimpled when the skin was moved. Fundi showed blurring of the disc margins. Roentgenograms showed separation of the sutures. Ventricular taps revealed enlargement of both lateral ventricles and a block between the ventricles and the spinal subarachnoid space. Following these diagnostic taps the block seemed to be temporarily relieved and the patient became alert and responsive. At this point a positive Babinski response could be demonstrated on the right. A cerebellar exploration was carried out and a firm, pale tumor of variable consistency exposed. It lay chiefly in the midline but extended somewhat into the left cerebellar hemisphere. In the depth of the tumor there were several long blonde hairs. The mass was completely removed.

Pathological Study. There were eleven fragments of tumor tissue available for examination, varying from $1.2 \times 1.0 \times 0.5$ cm. to $0.4 \times 0.3 \times 0.2$ cm. in overall dimensions. The portions representing cyst lining were smooth and grayish-pink in some parts and in others grayish-brown with strands of hair attached to the surface. The solid parts were gray or grayish-red with considerable variation in texture and appearance.

Histologically, the cyst was lined by rather coarse connective tissue with well formed hair follicles in the layer immediately subjacent. There was a rich infiltration with inflammatory cells and some multinucleated giant cells of foreign body type were in this region. These features were interpreted as reaction to keratinized cells, even though stratified squamous epithelium could not be demonstrated. Definite proof of the original character of the cyst lining was not obtained, and the conclusion as to its nature was based on the presence of hair, hair follicles and an inflammatory reaction similar to that seen in degenerated sebaceous cysts of the skin. The other parts of the tumor were composed of collagenous tissue with some infiltration by lymphocytes and mononuclear cells as well as polymorphonuclear leukocytes in a few

foci. There was considerable variation in degree of vascularity, but all blood vessels were essentially normal in structure. No other types of tissue were encountered. There was no evidence of invasion, dedifferentiation of cells or areas of rapid growth. Portions of adjacent brain tissue removed with the tumor showed extensive reactive gliosis.

Comment. This tumor was the only one in the series in which hair was demonstrated within the cyst at operation and the diagnosis made thereby without further notice. From the surgical point of view, the lesion was a simple, benign, cerebellar tumor. The patient remained well for ten years. She now has no objective residual changes but appears somewhat retarded.

CLINICAL ASPECTS—INTRASPINAL GROUP

As with the intracranial teratomas and teratoid tumors, the symptoms produced by the intraspinal members of the series were those of a space-occupying lesion at the level involved. With the intraspinal tumors, however, the association of a congenital abnormality was the rule rather than the exception (Cases 10, 12, 13, 14, and 15). Since these anomalies affected the vertebrae, they could be easily demonstrated roentgenologically and often by physical examination as well. The bony defects might or might not occur at the same level as the tumor. They were of considerable importance in suggesting the diagnosis of teratoma or teratoid tumor preoperatively. Usually the anomalies were compatible with normal function of the spine once the tumor had been removed and seldom required operative treatment. Occasionally, also, naevi or pigmented spots in the skin at the level of the lesion were regarded as somewhat suggestive of teratoma or teratoid tumor. None of these congenital abnormalities, when associated with signs of a space-occupying lesion, was conclusive proof of the nature of the lesion, but was merely suggestive. We have not regarded congenital anomalies of other organ systems, such as the cardiac or genito-urinary systems, as being in any way related to the incidence of the tumors under consideration.

Roentgenograms were chiefly useful in this group of patients for the demonstration of the presence and extent of an anomaly of the vertebral column. It is conceivable that a definite diagnosis might be made roentgenologically by the demonstration of calcified organ-like portions of the tumor. As in the intracranial group, no instance of this has occurred in the present series. Other roentgenographic studies for further localization and definition of the extent of the lesion were made as indicated as with any intraspinal tumor.

When the intraspinal teratomas and teratoid tumors were exposed at operation, they were found to vary greatly from one another. They might be extradural or intradural. The neoplasms were sometimes encapsulated and could be removed without injury to the spinal cord. The effects of these tumors on the spinal cord were rather like those of neurofibromas in similar locations. They were those of pressure and progressive interference with the vascular supply of the spinal cord. In other instances (e.g., Cases 10 and 11), a large cystic mass was found, the wall of which was not separable from the

spinal cord over a considerable area. These were treated by the evacuation of the cyst and removal of as much of the wall as could be carried out without damaging the nerve tracts further than had already been done by the tumor. In Cases 10 and 11, only a flat ribbon of spinal cord was left, which looked so seriously damaged that any return of function could hardly be antici-

FIG. 12. Case 9. A, exterior, and B, cut surface of a cystic intraspinal teratoma.

pated. In spite of the discouraging appearance of the spinal cord at the end of the operation in each patient, there was complete recovery of function in Case 10 and almost complete recovery in Case 11. Furthermore, the recovery has been maintained for ten years in Case 10 and four years in Case 11. Even though some of the cyst was certainly left behind in each patient, there has been no clinical evidence of recurrence at any time. This appears to be the most encouraging experience to emerge from the present study. No matter how discouraging a cystic intraspinal teratoma or teratoid tumor may appear when exposed at operation, it is in the best interests of the patient to evacuate all accessible cysts and to remove as much of the solid portion as can be accomplished without traumatizing the spinal cord. Even

though the final appearance of the operative field is such as would lead the surgeon to give a very grave prognosis if the lesion had been any other form of neoplasm, it is often possible for patients with partially removed teratomas and teratoid tumors to recover spinal cord function and live for many years—perhaps indefinitely—without clinical evidence of recurrence.

Of the 7 patients with intraspinal teratomas and teratoid tumors, 2 are

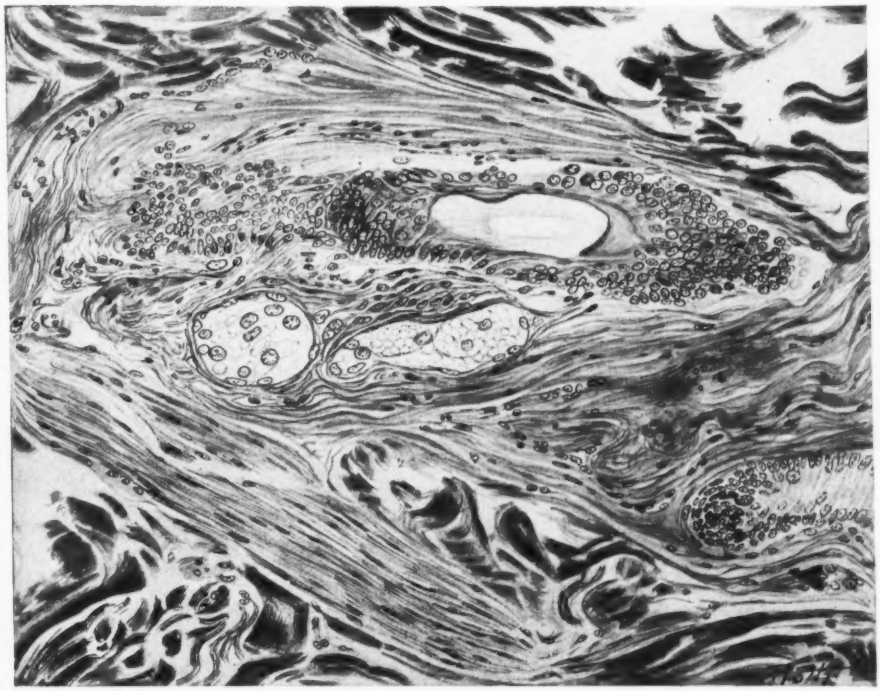

FIG. 13. Case 13. Camera lucida drawing of a hair follicle from a cystic intraspinal teratoma.

living more than five years; 4 are living for periods less than five years; and 1 is dead.

The following case reports are representative of the series of intraspinal cystic teratomas and teratoid tumors.

Abstract Case 10, C.S. (199355). This case has been reported in another publication.[4]

The patient was admitted to the hospital at the age of 2 5/12 years because of weakness of the arms and legs of 72 hours' duration. Seventeen days before admission she had been seen and treated for pain in the neck which appeared after a fall from the kitchen table. Roentgenograms made at that time showed absorption of the lamina of the 5th cervical vertebra interpreted as osteomyelitis. A Thomas collar had been applied for immobilization. At the time of entry she was acutely ill with flaccid paralysis of arms and legs with anesthesia except

over the shoulders and neck, where there was hyperesthesia. A diagnosis of compression of the cervical cord was made and laminectomy was done (Dr. R. U. Light). Lumbar puncture done at the time of operation revealed a complete block. A cystic mass was exposed, extending from the 2nd cervical to the 1st thoracic vertebrae. Ten to 12 cc. of yellow, gelatinous fluid were removed from the cyst and a nodule of firm tumor about 1½ cm. in diameter excised. This left the cervical portion of the cord merely a flat ribbon lying on the anterior wall of the spinal canal. Postoperatively there was rapid and complete return of function.

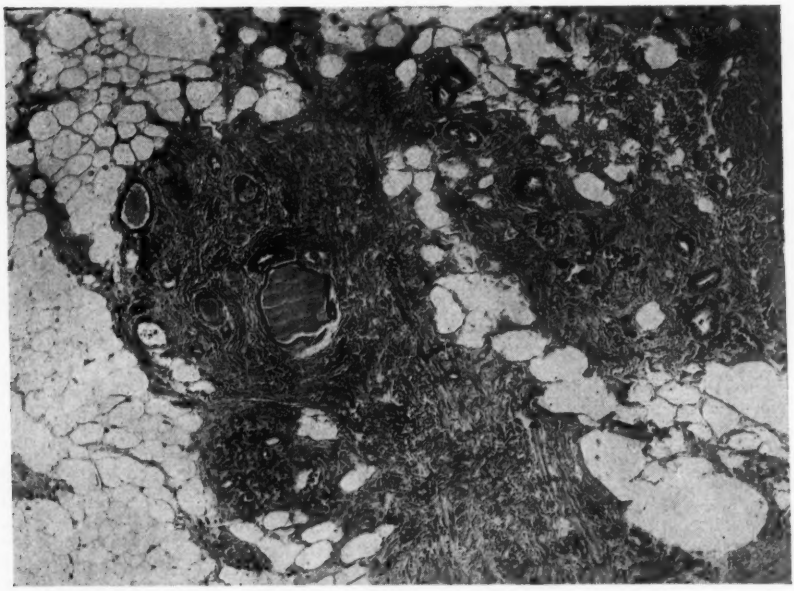

FIG. 14. Case 14. An area of fat, collagenous tissue and a few thyroid follicles with colloid from a cystic intraspinal teratoma. Hematoxylin and eosin stain, ×106.

Pathological Study. Two portions of solid tumor were available for study. One measured 1.2×1 cm.; it was smooth, moist, and dull pinkish-gray with streaks of red. There was a small cystic area in the center. The other measured 1×0.5 cm.; it appeared granular and was irregularly mottled pink and pinkish-yellow.

Histologically, the tumor was composed of several varieties of tissue. The large cyst was lined by pseudostratified ciliated columnar epithelium, which was thrown into folds in places and which formed crypts in others. Beneath the epithelium, there was a loose network of collagenous connective tissue interspersed with numerous mucous glands. The region, therefore, resembled somewhat portions of the respiratory tract. Other areas were composed of hyaline cartilage, bundles of nerve fibers, fat, and glands with large lumina lined by cuboidal epithelium. There were several regions filled with astrocytes and microglia. These were located at or near the periphery of the tumor and were regarded as reactive gliosis, but the possibility that some of them were an integral part of the tumor could not be entirely excluded. In the region of gliosis, the blood vessels had swollen endothelia and very thick adventitiae. There were no areas of rapid growth or invasion in any part of the material studied.

Section of a portion of dura removed from a region near the tumor showed congestion

as the only abnormality. A smear of the thick, slightly turbid, mucoid cyst fluid contained erythrocytes and lymphocytes in small numbers; no tumor cells were demonstrated.

Comment. The history of this patient illustrates the indolent growth and satisfactory clinical course of certain incompletely removed intraspinal teratomas. In spite of the fact that tumor was left behind at operation, there is still no evidence of recurrence after ten years. Histologically, the lesion was a teratoma without any areas of rapidly growing cells. The presence of ciliated epithelium relates it to the cases reported by Kubie and Fulton.[5]

Abstract Case 11, R.M. (262598). This patient was admitted with complaint of low back pain and difficulty in voiding. Examination showed a thin, rather undernourished boy in no distress. His spine was held rigidly fixed and both lower extremities were flaccid with marked atrophy and little muscle tone. There was hyperesthesia corresponding to the distribution of the 5th lumbar segment. Lumbar puncture done at the beginning of laminectomy showed a complete block with a very high protein. A cystic mass was exposed extending from the 1st lumbar segment to the lower end of the canal. The cyst was evacuated of thick brown, gelatinous material, and the wall and some solid tumor removed. It was impossible to do a complete extirpation without causing further damage to the lumbar cord. He was discharged home very much improved 21 days after operation.

Pathological Study. Studies were carried out on a portion of soft, friable, pinkish-red tissue $0.5 \times 0.3 \times 0.2$ cm. and a smaller fragment 0.1 in diameter.

Histologically, the tissue was composed of collagenous connective tissue of variable density in which were embedded discrete islets of keratinizing stratified squamous epithelium. In these regions, there was a large number of mucous glands and a moderate number of sebaceous glands but no hair follicles were present. There were a few areas of pseudostratified columnar epithelium. Fat cells were found in groups at numerous points. A smear made from the yellowish, mucilaginous cyst fluid showed erythrocytes and cellular debris. No keratinized cells were demonstrated.

Comment. Despite the extent of the lesion, the fact that tumor tissue was left at operation, and the severe compression of the spinal cord, this child is free from recurrence and has only very slight residual neurological abnormalities four years after operation. Histologically, the tumor was composed predominantly of keratinizing stratified squamous epithelium and skin appendages. It would, therefore, be regarded as a dermoid cyst had it not been for a few small groups of pseudostratified columnar epithelium. This is further evidence that dermoid cysts of the central nervous system should be considered with the teratomas and teratoid tumors. The presence of fat also links this tumor with the series described by Vonderahe and Niemer.[8]

SUMMARY

Fifteen instances of cystic teratoma and teratoid tumor of the central nervous system in children have been described. Eight of these were intracranial and 7 intraspinal.

All contained one or more cysts. The tumors contained tissue elements in their solid portions varying from only keratinizing epithelium to mixtures derived from all germ layers. One tumor, made up of undifferentiated nerve and striated muscle cells, was highly malignant, freely invading the cerebellum and seeding the meninges.

There was nothing to suggest the diagnosis preoperatively unless an associated congenital anomaly was present. The diagnosis could be established at operation by demonstration of hair, by finding keratinized or ciliated cells in the cyst fluids or by frozen section.

The use of adrenal cortical extract proved to be a valuable adjunct in an operation at which a teratoma of the third ventricle was removed.

Two of the patients are living five years or more after operation without evidence of recurrence; 5 for less than five years without evidence of recurrence; 2 have evidence of recurrence and 6 are dead.

In two patients, partial removal of intraspinal teratomas was followed by excellent return of function without recurrence of symptoms over a long period of years.

To say a patient has a teratoma or teratoid tumor of the central nervous system is no more indicative of the clinical result to be expected than to say the patient has a tumor. Some are simply removed, some are cured after a series of operations, some remain quiescent even though incompletely removed, some are so extensive that radical surgery is impossible, and some are invasive and malignant.

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THE EFFECTS OF SUBARACHNOID INSTILLATIONS OF THERAPEUTIC CONCENTRATIONS OF PENICILLIN SOLUTION ON THE CENTRAL NERVOUS SYSTEM*

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DURING the course of treatment of several patients with intraspinal instillations of penicillin in physiological salt solution, three cases were observed in which the spinal fluid became xanthochromic and evidently increased in viscosity. Also, the spinal fluid would jell or clot

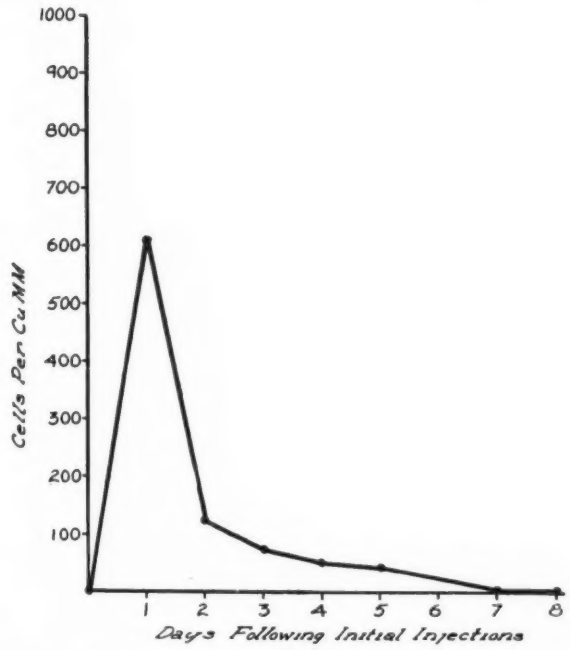

FIG. 1. Average daily cell counts following intracisternal instillation of a single dose of 1,000 units of penicillin (1,000 u/cc.).

* This work was begun with and partially supported by the Christine Breon Fund and completed under a contract, recommended by the Committee on Medical Research, between the Office of Scientific Research and Development and the University of California.

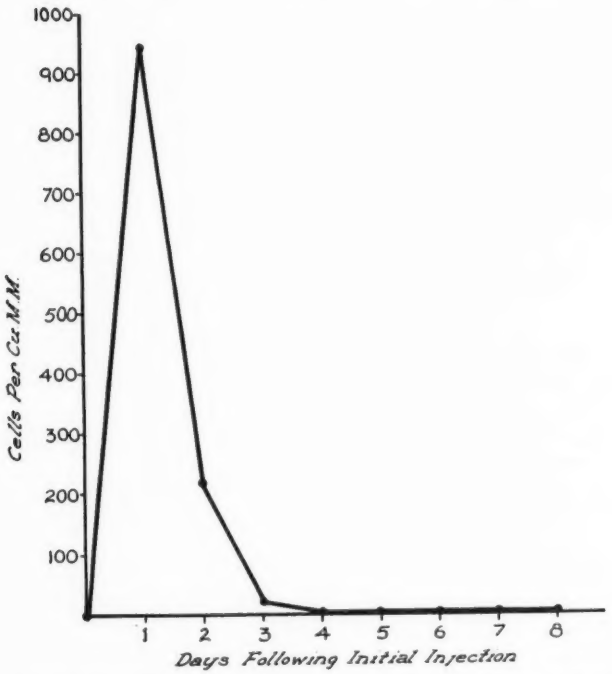

FIG. 2. Average daily cell counts following intracisternal instillation of a single dose of 2,000 units of penicillin (1,000 u/cc.).

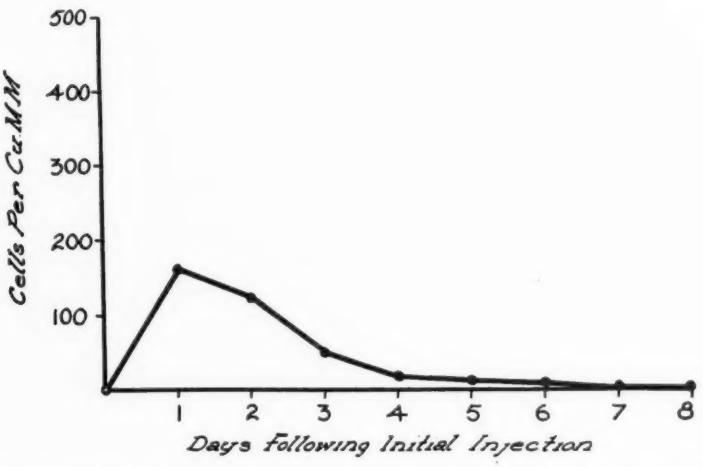

FIG. 3. Average daily cell counts following intracisternal instillation of 2 cc. of physiological salt solution.

within 20 to 60 seconds after it was removed from the subarachnoid space. These observations suggested the desirability of observing the effects of instillations of similar concentrations of penicillin in physiological saline into the cerebrospinal fluid of experimental animals.

Dogs weighing about 10 kg. were used. The amount of cerebrospinal fluid in these dogs was estimated to be about $\frac{1}{5}$ the amount in humans. The concentration of penicillin solutions used in these experiments was 1,000 units per cc. of physiological salt solution, the same as that used in the treatment of central nervous system infections in patients. Doses of 1,000

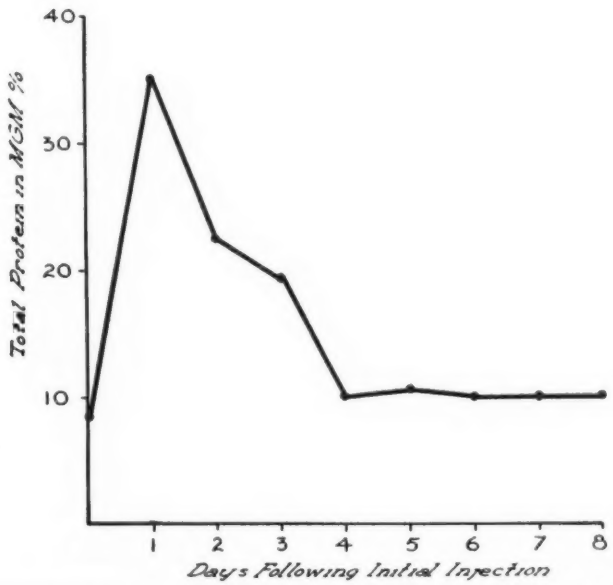

FIG. 4. Average cerebrospinal protein levels following intracisternal injection of 2,000 units of penicillin.

or 2,000 units of penicillin were used, corresponding to $\frac{1}{5}$ of the total dosage (5,000 to 10,000 units) that was being used in the treatment of human patients. The pH of the penicillin solution used in the experiments was measured and found to be 6.8, and the physiological saline used as control material also was pH 6.8. The animals were anesthetized with intravenous sodium pentobarbital immediately before each puncture. The rate of injection was made as uniform as possible by instilling the solution rapidly through a 19 gauge spinal needle, using very gentle pressure on the plunger of the 2 cc. syringe. Those animals in which bloody spinal fluid was obtained from cisternal or ventricular taps were discarded as unsuitable for the experiments.

In the first group of experiments a single instillation was made into the cisterna magna of 14 animals. One cc. of penicillin solution (1,000 units) was injected into each of 5 dogs, 2 cc. of penicillin solution (2,000 units) were injected into each of 4 dogs, and 2 cc. of physiological saline were injected into each of 5 control dogs. Subsequently, daily cisternal punctures were performed, and sufficient cerebrospinal fluid for cell counts and protein determinations was removed. A pleocytosis occurred in all animals, being lowest in the control animals that re-

ceived saline, and highest in the group of animals that received the larger doses of penicillin (Figs. 1, 2 and 3). The cerebrospinal-fluid proteins increased sharply during the first 24 hours after intracisternal penicillin instillation, and gradually returned to normal by the 4th day after injection (Fig. 4).

The animals were sacrificed 24 hours, 72 hours, 6 days, and 8 days after injection. The entire brain, spinal cord, and meninges were removed intact. Gross examination revealed no pathological findings, with the exception of small hematomas that occasionally occurred

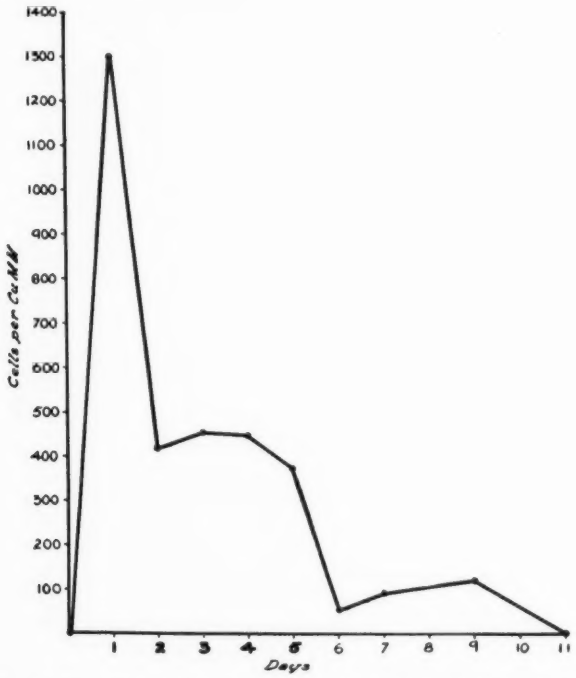

FIG. 5. Average daily cerebrospinal-fluid cell counts during and following intracisternal injection of 2,000 units of penicillin in 2 cc. of physiological salt solution on each of 5 successive days.

at the site of needle puncture. Microscopic examination made after fixing and staining these structures revealed no significant pathological findings.

In another group of 10 animals, 2,000 units of penicillin in 2 cc. of physiological salt solution were injected intracisternally into each of 5 animals on each of 5 successive days, and the other 5 animals served as controls, each receiving 2 cc. of physiological saline by cisternal injection on each of 5 successive days. Daily cerebrospinal-fluid cell counts and protein determinations were done and these were continued for 6 days following the final injections. The cerebrospinal-fluid cell counts (Fig. 5) and proteins increased sharply during the first 24 hours following the initial injection and gradually diminished during the succeeding days, even though the penicillin instillations were being given daily. Gross and microscopic examination of the brain, spinal cord, and meninges removed 11 days after beginning the experiment showed no significant pathological findings.

Penicillin solution (2,000 units in 2 cc. of physiological saline) was warmed to body tem-

perature immediately before the intracisternal instillation into 2 animals, and the pleocytosis and increase in cerebrospinal-fluid protein were found to be comparable to those observed in the other experiments. The administration of 2,000 units of purified penicillin dissolved in 2 cc. of the animal's cerebrospinal fluid also gave similar results.

No convulsions or other neurological signs developed in any of the animals receiving intrathecal penicillin. Electroencephalograms were made in a few of the animals that had received penicillin intrathecally, but no significant changes were observed.

Through burr holes, 2 experimental animals received a single intraventricular instillation of 2,000 units of penicillin in 2 cc. of physiological saline, and 1 animal received 2 cc. of physiological saline by the same route. Increases in the cerebrospinal-fluid cell count and protein were similar to those obtained by intracisternal instillations. No neurological reactions occurred and there were no significant pathological findings at necropsy.

Two cc. of penicillin solution (1,000 units per cc.) were instilled by lumbar puncture into each of 6 dogs. Spinal punctures on the days immediately following these penicillin instillations usually yielded only a few drops of yellow, gelatinous material, or were completely "dry." Cisternal punctures were performed 6 hours after lumbar intraspinal injection of 2,000 units of penicillin in 2 cc. of physiological saline in 3 of these animals. The cerebrospinal-fluid penicillin levels in these animals were 0.62, 0.15, and 0.00 units per cc. respectively.

COMMENT

In these experiments a temporary pleocytosis and increase in the cerebrospinal-fluid protein occurred following intrathecal administration of penicillin solution (1,000 units per cc.), but no abnormal neurological symptoms or signs appeared and no significant pathological changes were found. Before final conclusions are made it would be desirable to give lumbar intraspinal penicillin injections in a larger series of animals and make a more complete study of the protein and penicillin levels in the cerebrospinal fluid from the lumbar, cisternal, and ventricular areas, as well as necropsy examinations of the spinal cord and meninges in these areas. However, these experiments suggested that the increase in viscosity observed in the lumbar spinal fluid following intraspinal penicillin instillation is due to increase in the cerebrospinal-fluid protein together with the fact that there is a very slow diffusion of penicillin and cerebrospinal fluid from the lumbar region to the higher levels in the cerebrospinal-fluid system.

CASE REPORTS AND TECHNICAL NOTES

CEREBRAL GRANULOMA DUE TO SCHISTOSOMIASIS JAPONICA

A CASE REPORT

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Cerebral lesions due to Schistosomiasis Japonica are extremely rare. In a comprehensive review of this subject, Carroll[2] reported 5 cases occurring in army personnel on Leyte. He also reviewed the literature and found that only 8 previous cases had been reported. None of his cases was proven by either operation or autopsy and only 4 of those previously reported were so verified. Colonel J. E. Ash[1] of the Army Institute of Pathology in a personal communication stated that he had examined pathological material from the brain in 5 additional cases of Schistosomiasis in men returning from the South Pacific. Because of the fact that this condition has been so rare and yet will likely present itself in the future among returning army personnel, the following verified case of granuloma of the brain due to Schistosomiasis Japonica is reported.

CASE REPORT

The patient, a 33-year-old white male, was first seen Feb. 14, 1946 complaining of severe headaches and blurring of vision. He stated that he had only recently returned to this country from the South Pacific, having been discharged from the army in December 1945. He considered himself to be in good health until approximately 6 weeks previously, at which time he awakened with a severe frontal headache. Some 2 hours after onset of the headache he began to complain of blurring of vision without diplopia or hallucinations of vision, and then had a generalized convulsion. Thereafter, he complained of constant headache. He experienced only temporary relief following a lumbar puncture performed 1 week later. The spinal fluid was reported to be normal. Subsequently nausea and vomiting developed and in the 5th week of his illness, he apparently had one focal sensory seizure consisting of sudden paresthesias limited to the left arm. When seen at the end of 6 weeks, he was totally incapacitated with headache. The only additional symptom was mild incoordination and weakness of the left arm.

In his past history, several points of interest were elicited. He had been inducted into the army in 1941 and was sent to New Guinea in 1942. While there he had experienced three separate attacks of dengue fever from which he had recovered without complications or sequelae. Also while there, he had been temporarily incapacitated by an illness characterized by an abrupt onset of marked scrotal and penile edema, associated with fever, headache, malaise and mild abdominal pain. This attack lasted 4 days and was diagnosed as angioneurotic edema of unknown cause. It was not accompanied or followed by pedal edema. The patient was later transferred to the Philippines with his company of engineers and during the early portion of his stay on Leyte, he frequently waded and swam in streams and rivers while constructing bridges. During the interval from November 1944 to January 1945, he had three additional episodes of scrotal edema; each was similar to the first, and each lasted less than 5 days with no complications nor sequelae. In the last attack, however, the scrotal edema was accompanied by a marked edema of his lips and apparently of his pharynx as well. Each attack was preceded by fever and the last was ushered in by a chill. From the description of these attacks as submitted by the patient, it would be difficult to disprove the diagnosis of angioneurotic edema which was made on every occasion. A very interesting and pertinent point was made by the patient, when during the course of questioning, the possi-

bility was suggested that his present illness might be due to Schistosomiasis. He stated that he had seen several soldiers with this disease and that before he left Leyte, he had been subjected to stool and blood examinations to rule out this possibility. Furthermore, stool examinations had been made during the time of his last episode of scrotal edema. The patient was later transferred to Okinawa where he became ill with fever, malaise, jaundice, abdominal pain and loss of weight. A diagnosis of hepatitis was made and he was hospitalized for 2 months. He continued to exhibit mild jaundice as late as January 1946 but otherwise had no symptoms until the present illness.

Examination. The patient was a well developed white male who was obviously quite distressed by headache. There was no evidence of latent jaundice and the liver and spleen were

FIG. 1. Ventriculogram depicting the mass lesion in the right occipital region as evidenced by the anterior and superior displacement of the temporal and posterior horns of the lateral ventricle.

not enlarged. There was no evidence of edema of the scrotum and there was neither local nor generalized lymphadenopathy. The only positive findings upon neurological examination consisted of an early papilledema on the left and rather definite though slight hyperreflexia in the left extremities, without pathological reflexes. The perimetric fields and sensory studies were well within normal limits. X-rays of the skull revealed no noteworthy changes or helpful points of interest.

Course. With little more than a clinical "hunch" to support it, a presumptive diagnosis of Schistosomiasis was made and the patient was admitted to the hospital for further study. Because of the possibility of a parasitic infestation, repeated stool examinations were made but these yielded only ovae of *Endamoeba coli.* Blood studies were of particular interest in that there was a constant eosinophilia, on one occasion as high as 21 per cent. The white and red blood cell counts were normal, as were urinary studies. After a week of fruitless study and because the papilledema of the left nerve head seemed to be increasing, it was finally concluded that the diagnosis was incorrect. In order to rule out an intracranial lesion, ventriculography was advised and carried out. The ventricular air films revealed a mass lesion in the right occipital lobe (Fig. 1). The ventricular fluid contained 20 monocytes and 5,500 erythrocytes per c.mm.; protein content was 142 mg. per cent; Kahn reaction was negative.

Operation. A right occipitoparietal osteoplastic craniotomy was carried out following the ventriculogram. Other than for a somewhat vascular bone flap, no unusual pathology was encountered until the dura had been reflected. Upon visualization of the occipital cortex, there was at once evidence of markedly increased intracranial pressure, which was reduced somewhat by tapping of the posterior horn of the right lateral ventricle. The gyri were everywhere flattened and the cortex in general had an unusually soft consistency, so much so that one expected to encounter a subcortical cyst. However, on needling the cortex with a ventricular cannula, the consistency of the lobe was similar throughout with rather definitely decreased resistance encountered in all areas. After exploring the surface over the midline

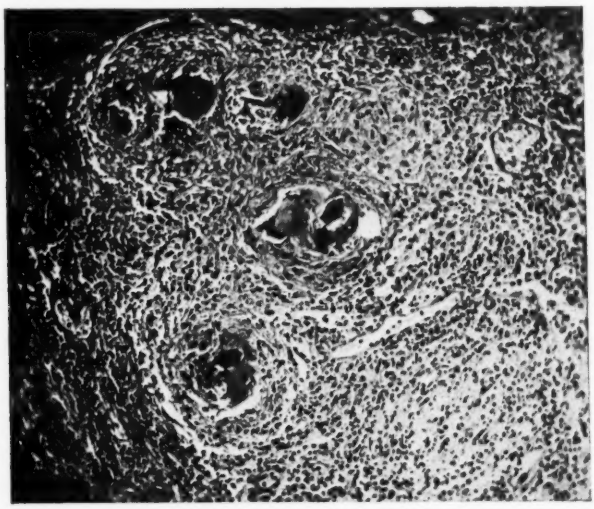

FIG. 2. Cerebral Schistosomiasis illustrating 4 partially confluent granulomata with prominent giant cells. The dark structures in the center of the granulomata are degenerated ova. (×100)

and finding no evidence of a surface lesion, the occipital pole was retracted anteriorly and as the calcarine area was visualized, an area of discoloration was encountered. This lesion measured roughly 3×3 cm. in diameter and was reddish-purple in color with markedly softened and gelatinous consistency. It proved to be an extremely elusive lesion, in that as one progressed inward, areas of softened but otherwise normal-appearing cortex alternated with rather obviously granulomatous tissue. When it became apparent that in order to carry out a gross removal of the lesion an extensive lobectomy would be required, we elected to sacrifice the entire occipital lobe as far forward as the posterior horn. The remaining cortex appeared uninvolved. The dura was sutured, the bone flap was wired in place and the wound closed without drainage.

Course. Postoperatively, the patient responded most satisfactorily, being immediately free of his formerly severe headaches and exhibiting only a complete left hemianopsia as a residual of the operative procedure. He was ambulant and without fever on the 6th postoperative day and discharged free of symptoms other than for the field defect, on the 10th day. In view of the indecision as to the nature of the granulomatous lesion, the patient was discharged before a definite pathological diagnosis could be established.

Microscopic Study. The sections show 3 fragments of cerebral cortex in 2 of which several large areas of inflammatory cell infiltration are seen. This inflammatory process involves the leptomeninges in the depths of the sulci with extension into the adjacent brain substance, and consists of numerous granulomata which often coalesce (Figs. 2 and 3). The granulomata are composed of large mononuclear cells with numerous, often huge, giant cells of foreign body type. In many instances these cells encircle one or several large parasites. Only ova, and no adult forms are seen. Most of the ova are in various stages of degeneration and show folding of the shell, loss of the cellular details of the enclosed miracidium and often calcification. There are, however, in these sections 2 well preserved ova which show an intact smooth shell and details of inner structure (Fig. 4). No spines are identified in these ova but

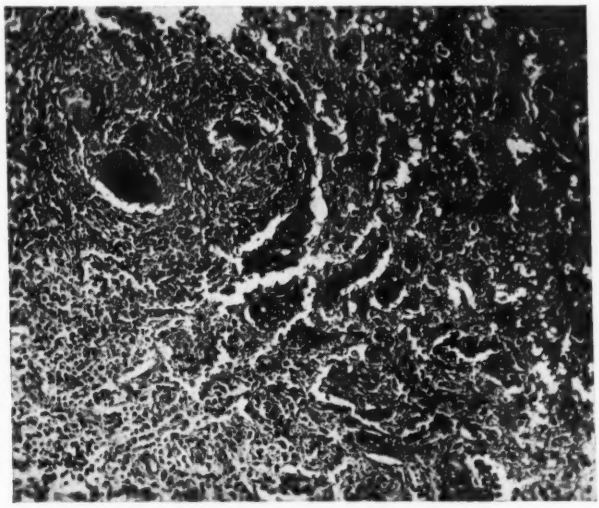

FIG. 3. Low power view of the granulomata and the adjacent cortex. (×100)

the lack of serial sections precludes a careful evaluation of this point. There is no caseation but occasional foci of recent central necrosis are seen. Neutrophilic polymorphonuclear leukocytes are present in these necrotic areas. The periphery of the granulomata displays numerous eosinophils as well as plasma cells and some lymphocytes. There is considerable proliferation from the leptomeninges of fibroblasts and capillaries. In the lesions situated in the brain substance proper, this proliferation originates from the perivascular connective tissue. The adjacent brain tissue shows edema and compression with varying stages of degeneration of ganglion cells as well as appreciable glial proliferation. Giant cytoplasmic astrocytes are numerous in these areas. The leptomeninges on the surface of the fragments show only slight infiltration by plasma cells, lymphocytes and large mononuclear cells. A similar infiltration is quite marked around the cerebral blood vessels even at some distance from the lesions.

In order to confirm or disprove our microscopic diagnosis and inasmuch as the author had not previously observed such a lesion, sections of this material were sent to Colonel J. E. Ash of the Army Institute of Pathology. Colonel Ash was of the opinion that the diagnosis of Schistosomiasis was well substantiated in this case and accordingly the patient's home physician was instructed to carry out the Faudin regime as used by the Naval Medical Corps. To date the patient remains asymptomatic.

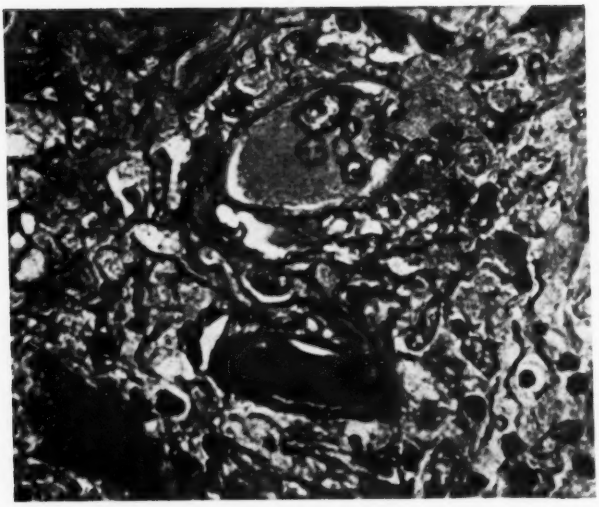

FIG. 4. A viable ovum of Schistosomiasis is seen in the upper part of the field. The structure beneath is a degenerated ovum. (×610)

CONCLUSION

A case of granuloma of the brain due to Schistosomiasis Japonicum is presented. It is to be pointed out that while this represents a rare situation, further cases will likely appear in the future presenting themselves as intracranial lesions.

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ARTERIO-VENOUS ANGIOMA (HAMARTOMA) OF THE BRAIN WITH INTRACEREBRAL HEMORRHAGE

REPORT OF A CASE WITH OPERATIVE REMOVAL OF THE HEMATOMA AND RECOVERY

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The arterio-venous angioma of the brain is a hamartomatous lesion composed of a tangled mass of interlacing vessels which resemble both arteries and veins.[1] These lesions have been described under a variety of names (Table 1) but in general have a rather characteristic morphology. When seen at the operating table they appear to involve little more than the surface of the brain, but at autopsy are seen as a wedge-shaped mass which extends to the ventricles. On the surface most of the vessels appear to lie in close proximity but deeper in the

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cerebral substance they are frequently separated by gliotic brain tissue.[5] Inasmuch as some portion of the angioma is in contact with the ventricle, rupture into the ventricular system during a convulsion is a common cause of death in patients with these lesions.[7] In the following case, however, the hemorrhage resulted in the formation of an intracerebral hematoma which was removed surgically.

The patient was a 38-year-old, obese male admitted to the North Unit of the Youngstown Hospital on Mar. 7, 1946. For about 2 years he had experienced intermittent headache associated with nausea and vomiting. There had been occasional twitching of the left side of the face and of the left upper extremity for about 1 year but these had not been constant nor had they been severe enough to alarm the patient.

The day prior to admission the patient had a severe headache with nausea and vomiting, and this was followed by what was described as a "fainting spell" with subsequent development of a spastic left hemiplegia. After this episode he became quite drowsy but continued to complain of severe right hemicranial headache. There had been no symptoms referable to the visual system and no evidence of a speech disorder. There was no history of tinnitus or

TABLE 1

Terminology	Author
Angioma arteriole	Cushing and Bailey[5]
Cirsoid aneurysm	Uiberall[13]
Arteriovenous angioma	Wolf and Brock[15]
Arterio-venous aneurysm	Bergstrand, *et al.*[1]
Aneurysmal angioma	Craig[2]
Arterio-venous hamartoma	Turner and Kernohan[12]

CASE REPORT

symptoms referable to the auditory system. The past history, aside from that given above, was non-contributory. Blood Kahn and Kline tests were negative; no abnormalities were found in the usual laboratory studies. The patient was right-handed.

Examination. He appeared to be acutely ill and quite lethargic but was nevertheless able to answer questions coherently. Blood pressure was 126/22; pulse 68; respirations 24; and temperature 99.2. There was a left spastic hemiplegia with practically total paralysis of the upper and lower extremities. Facial paralysis of central origin was present on the left and was severe enough to cause marked slurring of speech. However, there was no evidence of an aphasic disorder. The deep reflexes on the left were hyperactive and the Babinski sign was strongly positive on this side. The abdominal reflexes were absent on the left but normal on the right and there was sustained ankle clonus on the paralyzed side.

No sensory defect could be elicited on examination. Confrontation fields disclosed a left homonymous hemianopsia with sparing of the macular area as far as could be determined. The margins of the optic discs were blurred but there was no measurable elevation. The retinal vessels appeared hyperemic but otherwise normal. The left pupil was fixed and measured 9 mm. as compared to 7 mm. on the right. All other cranial nerves were normal. There was no bruit on auscultation of the skull. Roentgenogram of the skull disclosed no diagnostic abnormality.

Despite the absence of significant changes in the x-rays of the skull, the long history with few signs indicative of any marked increase in intracranial pressure over a 2-year period suggested the presence of a slowly growing tumor. However, the clinical evidence of extensive involvement, sufficient to include the leg, arm, and face area, as well as enough of the temporal lobe to involve the optic tracts, was rather difficult to visualize unless the lesion were situated deeply enough to include the internal capsule. Accordingly, ventriculography was carried out. On the left side the ventricle was reached at about 5 cm. from the surface and the

fluid, although under increased pressure, was normal in appearance. On the right, the ventricle could not be reached and no unusual resistance was encountered.

Films taken after the introduction of about 15 cc. of air into the left ventricle showed the ventricular system to be shifted to the left (Fig. 2). On the left side the entire ventricle could be outlined but on the right, only a small amount of air had entered the system, visualizing the posterior portion of the body, the occipital, and the temporal horns. There appeared to be a massive lesion occupying the region comparable to the anterior two-thirds of the body of the right ventricle.

Operation. Craniotomy was carried out under novocaine anesthesia and a large osteoplastic flap was reflected to include the posterior portion of the frontal as well as the upper temporal regions. The bone flap was centered over the motor cortex and the base of the flap was placed moderately low in the temporal region. The exposed dura was under moderate

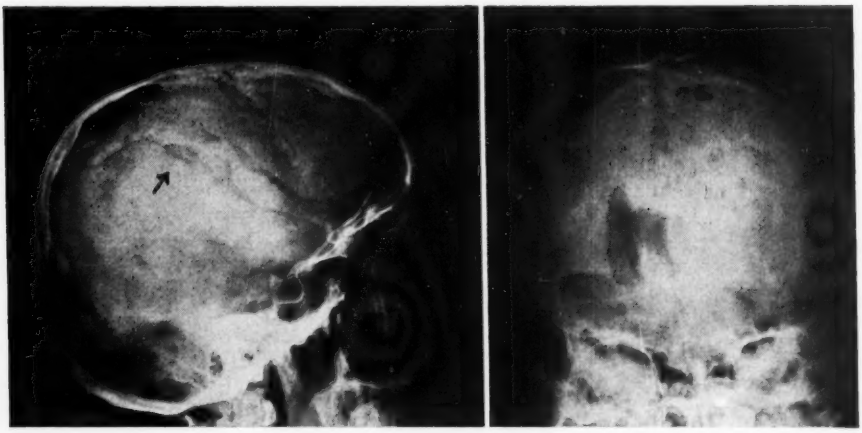

FIG. 1 (*left*). Lateral skull film taken preoperatively. Arrow points to small area of rarefaction which was found to correspond to the upper extent of the central dilated channel. Compare with Fig. 3.

FIG. 2 (*right*). Ventriculogram showing displacement of the ventricular system downward and to the left.

tension and in the central portion there could be seen a very large dark vessel which elevated this portion of the dura above the surrounding surface.

The dural flap was reflected with the base toward the superior longitudinal sinus. Upon opening the dura the brain bulged considerably and presented a remarkable appearance. The entire central portion of the exposure was covered by a mass of tortuous and coiled vessels which varied in diameter from 1 mm. or less to the largest, the exposed surface of which measured about 1 cm. in width (Fig. 3). The latter vessel corresponded to the position of the central vein and was partially buried in the cerebral substance. This vessel had a reddish-blue color, did not pulsate visibly, and followed the course of the central fissure to disappear into the substance of the brain just short of the upper edge of the exposure. Proximally it disappeared into the lip of the Sylvian fissure. The individual vessels varied in appearance between that of arteries and veins but about two-thirds of those composing the mass were of a peculiar reddish-blue color. A few of the vessels pulsated noticeably but as a whole the lesion did not show the degree of pulsation that is usually present in the typical arterial type of angioma. In the central portion of the lesion the cortex was completely covered by the mass of vessels but at the periphery the vessels were slightly smaller, somewhat less closely applied, and the adjacent cortex had a fine capillary network on the surface.

Just anterior to the lip of the Sylvian fissure was an area measuring approximately 2 cm.

in diameter which was the site of vascular thrombosis and scarring. The pia-arachnoid was thickened and had a gray-white color with considerable yellow and brown pigment present. This area was moderately firm to palpation and had the appearance of a lesion that had been present for some time. A similar area was present just anterior to the upper third of the

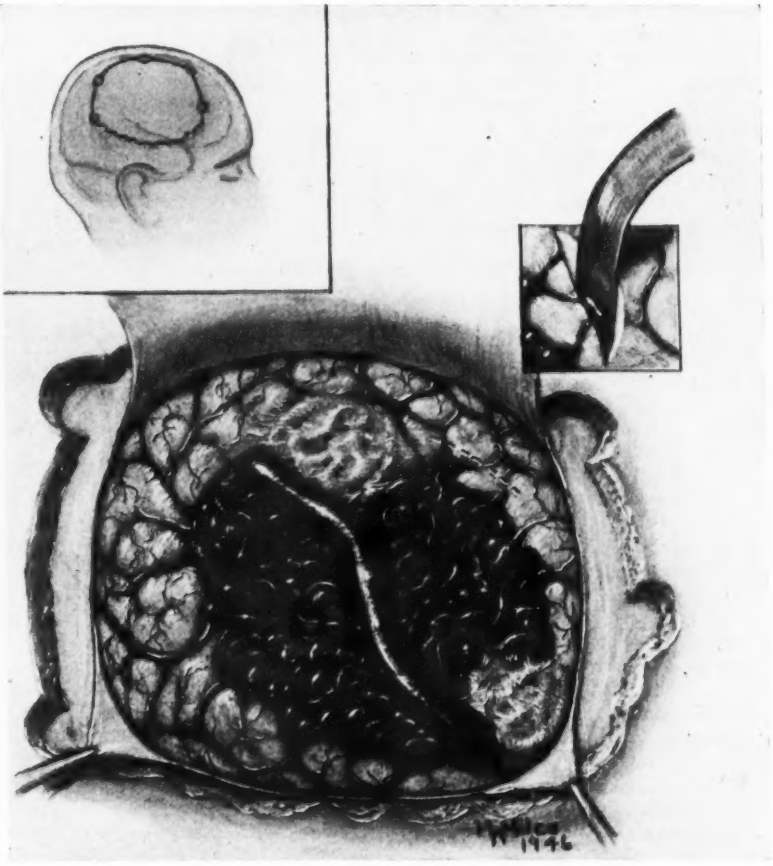

FIG. 3. Reconstructed drawing of lesion found at operation. Note the dilated central channel, the variable character of the vessels and the two areas of thrombosis. Dotted line indicates site of cortical incision. Inserts show site of bone flap and method of exposing hematoma.

enlarged central vein and involved the vessels of the varix at the anterior edge of the tumor mass.

The brain itself appeared to be moderately soft and palpation disclosed no firm tumor, nor did the introduction of a ventricular needle at the periphery of the lesion locate an area of abnormal resistance. Because the degree of herniation of brain appeared to be greater than could be accounted for by the varix itself, a small incision was made in the cortex at the anterior and superior edge of the varix. At a depth of about 2–3 cm. a large currant-jelly type of clot was encountered; about 50–60 cc. of this material were removed. The remaining cavity

appeared to underlie the upper two-thirds of the motor cortex and biopsy of the wall disclosed no evidence of tumor. Following removal of the clot there was considerable hemorrhage from an area just under the mid portion of the varix and this required the use of a considerable amount of fibrin foam to control. Nothing was done to the varix itself and the wound was closed allowing a large decompression in the temporal region.

Postoperative Course. This was uneventful. Eight days after operation the patient had slight voluntary movement in the upper extremity and this progressed slowly. He was discharged from the hospital on the 15th postoperative day.

When seen approximately 6 months later the area of decompression was soft and pulsated quite markedly. For the first time a loud systolic bruit could be heard on auscultation of the operative site as well as in the right carotid region. The optic discs showed no elevation but the margins were moderately blurred. The retinal vessels appeared to be slightly distended but there was no evidence of a vascular tumor of the retina. There was a moderate degree of voluntary movement in the right lower extremity, but the improvement in the function of the right upper extremity had not progressed very far.

DISCUSSION

The complete clinical syndrome of the arterio-venous angioma as described by Cushing and Bailey[5] consists of (1) intracranial bruit; (2) increased extracranial vascularity (vascular scalp, carotid hypertrophy, and cardiac hypertrophy); (3) papilledema; and (4) convulsive seizures often with focal symptoms and frequently with long periods of remission. However, as Noran[7] has pointed out, intracranial bruit and increased intracranial pressure are not common findings. It must be remembered that Cushing and Bailey[5] emphasized this variable feature of the bruit.

Inasmuch as the vessels forming the lesion are closely related to or partially composed of those vessels that normally supply the brain, interference with many of these vessels may lead to infarction of additional cortex. Also, indiscriminate closure of venous channels draining the mass of vessels may lead to a considerable retrograde distension of the deeper veins with subsequent rupture of the overlying cortex or further impairment of circulation to the brain substance.

In addition to the symptoms listed above, these lesions may cause acute manifestations by several means. Subarachnoid hemorrhage, such as described by Noran,[7] may occur in association with or possibly as the prodromal phase of an intracerebral or intraventricular hemorrhage. Intrinsic thrombosis with subsequent further impairment of the peripheral circulation may result in the occurrence of focal symptoms while, as has been noted above, massive hemorrhage may precipitate acute intracranial pressure. In the case described above, the relation of the thrombosis of portions of the lesion to the history of focal twitchings involving the left side of the face and left upper extremity is not clear.

It has been pointed out that massive hemorrhage into the ventricular system is the usual cause of death in a patient suffering from this type of lesion, but the case described here illustrates the fact that extensive hemorrhage is not necessarily followed by fatality and that rupture may occur into the substance of the brain rather than into the ventricular system. According to Craig and Adson,[3] Cushing was probably the first to actually remove an intracerebral clot. Since his report in 1903[4] a considerable literature has grown with many reports of successful evacuation of an intracerebral hematoma. A perusal of the literature, however, reveals no instance in which the hematoma was secondary to an angiomatous lesion. In Cushing's case[4] the hemorrhage was secondary to hypertension and although the patient survived the operation he died 2 weeks later from pneumonia. Russell and Sargent[10] reported a case in 1909 and here again the hemorrhage was apoplectiform. Most of the intracerebral hematomas described in the literature can be placed into one of three etiological groups as follows: (1) post-traumatic, immediate or delayed; (2) secondary to hypertension and/or arteriosclerotic cardiovascular disease; and (3) of unknown etiology. Naffziger and Jones[5] reported 3 cases in 1928, all being of the delayed post-traumatic type. Penfield[8] in 1933 reported 2 cases, one of a young boy in whom the hemorrhage occurred in relation to excessive

vomiting following smoking. In the second patient, the hemorrhage was apparently due to hypertension.

Pilcher[9] has recently reported 8 cases of hematoma, 3 of which were post-traumatic and 5 of unknown etiology. Of the latter, one patient was known to have arteriosclerotic heart disease. Craig and Adson[3] in 1936 reported 9 cases of operative evacuation of an intracerebral hematoma. Of these, 2 patients had a definite history of trauma, 1 developed symptoms following a week-end of strenuous activity, and a 4th, with symptoms of 4 months duration, had had an injury 12 years previous to operation. In 4 other cases the cause of the hemorrhage was unknown, while in still another the hemorrhage was probably related to rheumatic fever and malignant endocarditis. Wilkins'[11] two cases were both of traumatic origin.

In most of the cases reported recovery has been the rule and when death has occurred following operation it has usually been the result of complications or related to the etiological condition rather than the hemorrhage itself. Pilcher[9] reported 2 deaths: one patient was a 62-year-old man who died of unknown cause 6 months after operation; the other was a 56-year-old man with arteriosclerotic heart disease, who had recurrence of symptoms 2 months after operation and died 7 months later. Of the group reported by Craig and Adson[3] one patient died on the 9th postoperative day, possibly from cerebral hemorrhage, and the other died of endocarditis 3 months after operation.

It is to be noted that removal of the hematoma in the case described here was followed immediately by severe bleeding, apparently from the original site of hemorrhage. It seems likely that the pressure developed by the hematoma itself was sufficient to occlude the bleeding vessel, preventing massive hemorrhage with subsequent death. In retrospect, examination of the x-rays of the skull showed an area of bone absorption which corresponded with the upper portion of the large central vessel in the tumor-mass (Fig. 1). However, there were no changes in the skull to suggest the presence of a vascular lesion.

SUMMARY

A case has been described in which an intracerebral hematoma resulting from rupture of a deep vessel of an arterio-venous angioma (hamartoma) was removed surgically.

It has been pointed out that acute symptoms of intracranial pressure in the presence of a vascular lesion may be caused in several ways and that intracerebral hemorrhage may not necessarily prove fatal.

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A NEW METHOD FOR THE CONTROL OF HEMORRHAGE IN CEREBRAL SURGERY

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"All surgeons who make for themselves opportunities to observe the manipulative work of their fellows must appreciate the present tendency toward the abandonment of the applauded methods of comparatively few years ago." With this statement Dr. Harvey Cushing in 1911 introduced a paper on the control of bleeding in operations for brain tumors read before the American Surgical Association.[1]

In the field of neurological surgery, few if any useful methods have been relegated to the scrap heap and many neurological surgeons, during the course of a single operation, may make use of all possible aids to control hemostasis from the scalp incision until the last suture has been tied.

In no other type of surgery does bleeding so obscure the clear view essential to the safety of delicate manipulations as are required in surgery of the central nervous system, unless it is cardiac surgery. White's observations have disclosed that the loss of blood in major neurosurgical operations is much larger than had been suspected, and his estimates of the amount lost, from 1 to 2 liters, were confirmed by Webster at the University of Pennsylvania.[3,4]

In order to minimize the loss of blood, the neurological surgeon has summoned to his aid numerous useful methods. Among them are hemostasis with various clamps and tourniquets, the application of weights on pendent instruments thus allowing the galea by mere weight to close the bleeding points, the use of novocain fortified with epinephrine, bone wax to stop bleeding from diploic spaces of bone, muscle fragments, cottonoid pledgets and strips, silver and tantalum wire clips, gauze, ligatures, hot irrigating solutions, gelatine sponge, thrombin, electro-coagulation and suction.[2]

With all these methods ready at hand the neurological surgeon's ability occasionally remains challenged by an unsatisfactory anaesthesia. Notwithstanding the use of all these devices for the control of hemorrhage, an occasional case may be encountered in which all fail and consequently prolong the operation. To overcome this a new device has been developed which has been tried out by Dr. Sachs and is now being used on the neurosurgical service at Barnes Hospital.

An apparatus of simple construction has been designed to speed up a clear-view exposure of bleeding points so that electro-coagulation can be rapidly performed, and at the same time eliminate many cumbersome, obscuring and often elusive cottonoid strips. The method makes use of a controlled jet of sterile compressed air delivered at the bleeding site, permitting precise exposure of severed vessels for electro-coagulation. The apparatus consists of a series of bottles containing antiseptic solutions to wash the compressed air, and the conventional sucker tip is used for its delivery. The volume of air pressure is controlled by a flow-meter which delivers 12 to 15 liters per minute.

We have found that the most effective way to use this is for the surgeon to hold the jet tip in his left hand and direct the jet at the bleeding point and at the same time use his forceps with his right hand to grasp and coagulate the severed vessel. The assistant performs his duties in the usual manner by suctioning the excessive blood from the general area without compromising the vessel point and its exposure. This procedure we have found reduces the necessity of frequent cottonoid packing, irrigating, blind hazardous sucking and electro-coagulation.

Use of the method has shown in many instances hemorrhage that was thought to come from generalized capillary oozing actually comes from a single bleeding point, which the jet will expose and which then can be accurately and precisely coagulated. Surgeons who perform major secondary neurosurgical exposures on their own patients and referred patients,

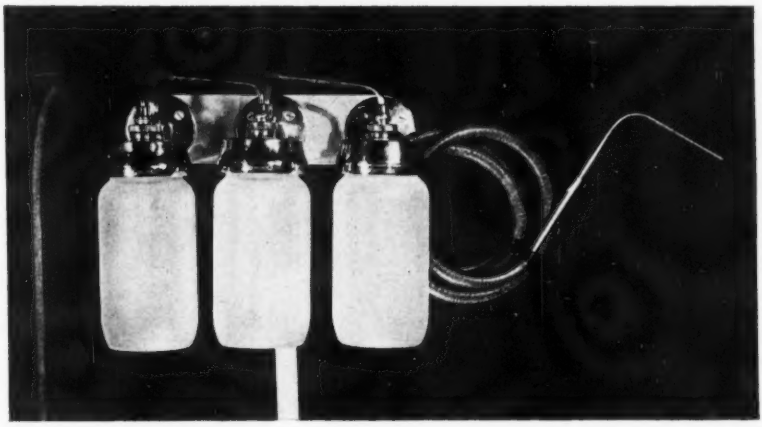

FIG. 1. Apparatus used for sterilization and delivery of compressed air.

realize the difficulties produced by a field in which muscle stamps, indiscreetly placed, have produced excessive scarring at secondary operations, whereas the electro-coagulated areas are glistening and well defined.

Even surgeons with assistants who are capable of constantly keeping the field clear in which there is excessive bleeding labor under a false security which would be considerably shaken if they had the blood-filled suction bottles placed before them with the piles of blood-soaked cottonoid that accumulate during an operation.

The device here described has helped us to minimize the loss of blood and has simplified and shortened the length of neurosurgical procedures, and for this reason it is presented as a further aid in neurosurgical technique.

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A SPONGE RUBBER PLASTER COLLAR

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In the Naval Service where large numbers of cervical spine fractures, both with and without cord or root damage, were treated, we were faced with the problem of post-reduction immobilization. The usual methods of attaining this left much to be desired in that they were not readily adaptable or were too heavy and therefore uncomfortable for the patient. The advantages of the collar described below are that it is individualized, comfortable, and can be manipulated by the patient. In addition it is simple and inexpensive.

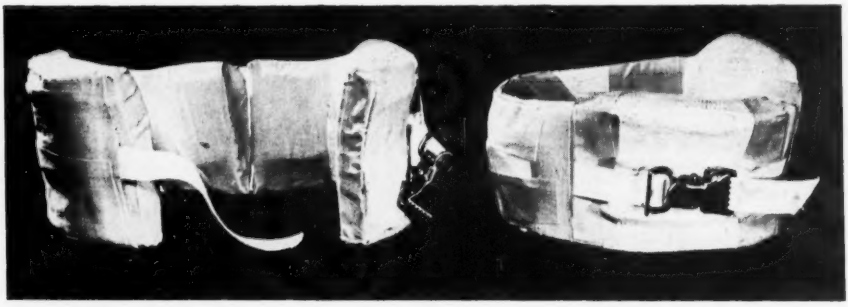

FIG. 1

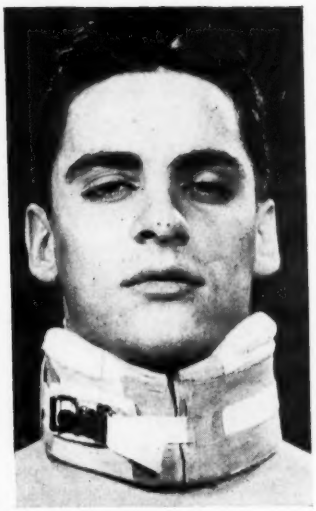

FIG. 2

Stockinette of sufficient diameter and length is slipped over the head and around the neck while traction is maintained. It must not be wrinkled. Sponge rubber sheeting ($\frac{3}{4}$ inch) is cut to the size and shape of the collar required. A single roll of plaster is wound around the sponge rubber. The upper and lower edges of stockinette are incorporated in the last few turns. Two Gigli saws which have been encased in drinking straws are then passed between the collar and the skin. These are used to bivalve the collar when it has set. The two halves can then be hinged posteriorly by means of adhesive. A web tourniquet is then fastened to the collar. This with its spring clip makes a simple fastener. Shoulder and occipital supports can be made in the original mold if required.

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HARVEY CUSHING. A BIOGRAPHY. By John F. Fulton, M.D. *Springfield, Ill.: Charles C Thomas*, 1946, xii, 754 pp. [150 illustrations]. Price, $5.

Dr. Oliver Wendell Holmes once said, "Think what a horrid business the biographers make of a man's private history!" Dr. Cushing may often have thought of this warning during the months that he worked on *The Life of Sir William Osler* and during the years after its publication when he realized how inspiring to many people, and particularly to young doctors, may be the well-written record of a great physician. In any event, four years before his death he determined to leave by will to Mrs. Cushing and his literary executor the decision as to whether after his decease an account of his own life promised to be of sufficient interest and help to medical students as to justify its writing and publication; and in order to make the writer's work as easy as possible—should a writer ever be chosen for the task—a peculiarly well-documented description of his experiences during the seventy-year span of his existence.

Thus Dr. Fulton's *Harvey Cushing* is an unusual biography: in many ways it is an autobiography written by Dr. Cushing with Dr. Fulton's editorial assistance, rather than a more impersonal view of his life as seen through outside eyes. That the book is so largely autobiographical makes it all the more interesting.

The volume begins with a sympathetic narrative of Dr. Cushing's ancestry and early days, revealing not only how strong were his roots in medical tradition, but also how deeply were they planted in Cleveland, the Western Reserve, and Yale University. One gets the picture of an alert-minded, attractive boy, well brought up in a hard-working, conscientious doctor's family, living in a relatively small community, surrounded by brothers and sisters, cousins and friends, and endowed with winning manners, a love of nature, great energy, a sense of humor, an interest in collecting, and with a pen which proved to have a knack at picturesque expression almost as soon as it could form words.

From such a beginning Dr. Cushing's development to full maturity is carefully traced year by year, almost day by day; through Yale College, the Harvard Medical School, the Massachusetts General Hospital, the Johns Hopkins—where Osler admonished him in the words of the Psalmist: "Keep your mouth shut as it were with a bridle"—abroad, and finally once again to Boston where he remained for twenty years as Professor of Surgery at Harvard and Surgeon-in-Chief to the Peter Bent Brigham Hospital.

The final chapters deal with Dr. Cushing's later days when the time had come for him to retire from Harvard and return to Yale. These chapters emphasize how he continued until the end to have an unquenchable interest in books, young students, old friends, and new medical projects; above all, in Yale and in the careers of the men whom he had trained and who owed so much of their ultimate success to his influence.

Dr. Fulton has shown rare judgment in selecting for publication mainly that material which gives Dr. Cushing fullest opportunity to paint his own portrait. The growth of his surgical skill, his fame, his ingenuity in research, and his ability to write; the people he encountered; his friends, his avocations, his travels; the decisions he was forced to make and how he made them; his views on life, his artistic talents, his ideals, his disappointments; his impatience, his patriotism, his honors; the unique charm of his personality; his ever broadening interests and influence—all these, and more too, are brought out through the medium of letters or diaries. In brief, the book gives the reader a delightfully personal and vivid description of all kinds of happenings in a doctor's life as they took place and influenced its course during an astonishingly variegated and full lifetime.

A biography of such character will attract many different groups of readers—medical students for whose benefit, especially, Dr. Cushing hoped it might be prepared; physicians; historians who will use it as a source of reference; and, indeed, writers. After reading it, a great many persons are certain to wish to say to Dr. Fulton about this book, almost exactly what Mr. William H. Taft said to Dr. Cushing about *The Life of Sir William Osler:* "I finished it

last night with moist eyes. . . . The preparation of such a biography must have involved the greatest labor. . . . You have erected a monumental work of highest value. . . ."

REGINALD FITZ

VICTORY OVER PAIN. A HISTORY OF ANESTHESIA. By Victor Robinson, M.D. *New York: Henry Schuman*, 1946, xiv, 338 pp. [47 illustrations]. Price, $3.50.

Victory Over Pain is an absorbing historical survey of the development of agents for the alleviation of pain. The events leading to the discovery of ether and other anesthetics are so vividly described that one can scarcely tolerate an interruption to its reading. Fortunately it skillfully avoids most of the pitfalls of books by or about scientists. It is neither pedantic nor labored and is free of undue dramatization.

The book gives a moving narrative of the battle against pain associated with surgical procedures and childbirth. Its arrangement is essentially chronological, but it necessarily hits only the highlights of the history of anesthesia. The use of narcotics and hypnotics during ancient and medieval times and in the Orient is described in about forty pages. Many other sections of the book are regrettably short. In the description of medicine in Edinburgh, whole storehouses of interesting information had to be reduced to fragmentary size. These comments are made with full knowledge that the author had to limit the size of the book and yet was obliged to cover such a large field. It is to be hoped that he will eventually write in greater detail about these events.

There is an admirable balance between the body of the narrative itself and a running ornament of interesting biography. The reader becomes acquainted with Sir Humphry Davy, Thomas Beddoes, John Elliotson, Crawford Long, Horace Wells, Charles T. Jackson, William Morton, James Syme, Nicolai Pirogoff, Sir James Simpson, Samuel Guthrie, John Snow the first specialist in anesthesia, and others who paved the way for modern inhalation anesthesia.

An honestly interpretative light is thrown on the weaknesses as well as the virtues of the men involved in the historical events. The pride and inapproachability of Sir Humphry Davy in his later years that made him inaccessible to Henry Hickman and probably postponed by more than twenty years the acceptance of the idea of anesthesia by inhalation of gases; the paranoid tendencies of Charles T. Jackson; the weaknesses for publicity on the part of William Morton; and the failure of other men, even though they were keen observers in some respects, to see what they had before them, as in the case of Samuel Guthrie who observed the action of chloroform without grasping its importance.

The underlying motif of the book seems to be the importance of basic and conservative research. Much as the early surgeons would have liked to spare their patients pain, it was not possible until the labor of "pure sciences" bore fruit. Until the alkaloids were separated and prepared in relatively pure form, dosage in the administration of opiates was fortuitous, and successful therapeutic results were obtained with difficulty. The careful isolation of cocaine, the synthesis of non-habituating procaine, and the quite recent development of divinyl ether, based upon the chemist's ability to predict behavior of certain molecular configurations, are examples of the laborious footwork that must go into the forward steps in scientific achievement.

In addition to the account of the use of carbon dioxide, nitrous oxide and later ether, culminating in the first public demonstration of surgical anesthesia at the Massachusetts General Hospital one hundred years ago, the book also describes the history of the development of chloroform for childbirth, the first tragic investigations with cocaine, the development of the barbiturates and other anodynes. Techniques of rectal, spinal and caudal anesthesia, and of anesthesia by refrigeration, and even the addition of curare to the anesthetist's armamentarium are well oriented to the pattern of this history.

One is impressed by Victor Robinson's ability to describe complicated physiological processes in a way so that neither rhetoric nor medical concepts are violated. The book is well

illustrated by a number of unusual and old prints, and there are some charming line drawings of plants used in ancient therapeutics. There is also a brief list of selected and annotated references. *Victory Over Pain* will make a very useful addition to a medical library, for it cuts a broad and accurate swath in the complicated history of anesthesia.

ROBERT B. LIVINGSTON

THE CENTENNIAL OF SURGICAL ANESTHESIA. An Annotated Catalogue of Books and Pamphlets Bearing on the Early History of Surgical Anesthesia. Compiled by John F. Fulton, M.D., and Madeline E. Stanton, A.B. *New York: Henry Schuman*, 1946, xvi, 102 pp. [9 illustrations]. Price, $4.

This is Publication 15 of the Historical Library of Yale Medical Library. The catalogue is a scholarly work in which selected significant papers and other communications on the early history of anesthesia are brought together. The present publication constitutes Part I of the catalogue, and is to be followed by a second part on regional and block anesthesia, and other anesthetics after 1875. The catalogue is attractively arranged for easy reference without obstructing reading, and a number of old title pages are reproduced together with a striking portrait of William T. G. Morton. Each section is followed by a list of references to biography and commentary, and a list of journal articles and monographs completes the work.

ROBERT B. LIVINGSTON

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